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NEW YORK CITY HOSPITAL

**MEDICAL AND SURGICAL
REPORTS**

VOLUME II

1915



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INTRODUCTION

Since the last Medical and Surgical Report of the Hospital was published many important changes relative to the Executive Management of the Hospital and various Divisions have been instituted which seem worthy of more than passing mention.

The appointment of a Medical Superintendent and Executive Intern has contributed greatly to the harmony between the Medical Board, Interns, and Commissioners, and has added greatly to the general efficiency of the hospital service.

The reorganization of the Pathological Laboratory, which was effected in June, 1911, has resulted in a closer association of the work of the pathologist to the clinicians in the Hospital proper, and has added greatly to the general efficiency of all divisions. For some years previous the Pathological Laboratory was maintained by a private endowment, but it was thought best that this connection be discontinued, and, accordingly, a change was brought about which resulted in the appointment of a pathological staff to be paid by the city, such workers being Civil Service appointees directly under the supervision of the Charities Department. A Director of Pathological Laboratories was appointed with three assistant pathologists from the Civil Service lists. This was the first step toward the complete reorganization of the then existing conditions, and the placing of the Pathological Laboratory on a modern basis, so that the greatest amount of good might result to the clinicians and, in turn, to their patients in the wards.

It is hardly necessary to say that such a change has resulted in much benefit to the hospital and to the clinicians and the patients under their care. It was found that the building given to the city by Emma Strecker was in a bad and unsanitary condition. The building contained no apparatus of a modern character, not even proper lighting facilities or electrical appliances. Consequently it had to be completely equipped to do the work required of a well-conducted laboratory. The building was completely renovated, and many interior changes made. A complete stock of modern laboratory apparatus, chemicals, stains, glassware, etc., was installed.

The Strecker Memorial Laboratory as it now stands is a three-story stone building, situated in close proximity to the Hospital, on the east shore of the island. The first floor comprises the Autopsy and Lecture Room, a room for Pathological Chemistry, one for Serology, and another for pathological museum specimens.

The second floor is subdivided into two rooms: one a general room in connection with the media-making; the other a room set aside for section

work, with suitable containers for instruments and glassware. A small but well-supplied stock room closely adjoins, containing all the recent stains used in modern laboratories.

The top floor consists of three rooms, one of which is set aside for the use of the Director, which contains the pathological library and tables for special workers. Adjoining this is the Record Room, and next is a room for general Bacteriology.



Janeway Hall—Resident Intern Staff House.

The character of work done in the laboratory is thoroughly up to date:

General and special pathological work on all material coming directly from the wards of the Hospital, systematic autopsy work, and the recording and reporting of the findings in such cases.

A new Department of Pathological Chemistry has been established, and the room set aside for this purpose is well equipped with all the necessary apparatus, and is well stocked with all the chemicals and reagents necessary for conducting this branch of work. This department is in charge of a pathological chemist under the direction of the Director. No well-con-

ducted laboratory at the present time can succeed or keep abreast of modern medical thought without such an equipment, and during the past two years, since the establishment of this department on a modern basis, a vast amount of work has been accomplished that has resulted in direct benefit to clinicians and patients alike.

The Pathological Chemist makes chemical examinations of material sent to the laboratory by the clinicians from the wards of the Hospital, and all



Superintendent's Cottage.

special examinations of material preserved at autopsy on bodies showing the rarer forms of disease. During the past year a number of important chemical examinations of a special character have been made with the object of elucidating the causative factors underlying such diseases.

The work done by the Department of Serology has been of great value. Perhaps no hospital in the City of New York commands such a variety of cases to which special work might be applied with greater advantage than the City Hospital. Upward of from 6,000 to 7,000 individual reactions have been made during the past year, and during the past two years some

15,000 individual examinations of a serological nature have been completed. Numerous changes in the character of the technique used in the work have been made from time to time, with a view to keeping abreast of all advances made in this field. The publication of the work and the keeping of records in this department have been maintained with scrupulous care, and already a sufficiently large amount of data has been collected. This will be made the basis of a later report by the Director.



New Reception Office.

The Bacteriological Department during the past year has been maintained on an equal footing with the other departments. This department now has in stock all of the well-known bacteria used for special work. A full stock of all special media is constantly kept on hand for the use of the laboratory workers, as well as the clinicians of the Hospital. During the past year the very large bacteriological service has been accurately done; the preparation of all vaccines, the making of all special bacteriological examinations, and the recording and filing of all such data and the prompt report to the wards have been made possible.

The systematized instruction now given to the interns by this department is of incalculable value to them, and is only one of the many benefits which the hospital is daily deriving from the hard and unselfish work of its Pathological Director.

Since the establishment of the 70th Street Relief Station, the influx of patients to the City Hospital has been very materially increased, and the Ambulance Service now brings in many acute cases, which previously were



Autopsy and Lecture Room.

not admitted to the Hospital, and which have added greatly to the training of the interns and nurses. Additional sources of patients are from 26th Street, and from the Visiting Physicians of the Hospital and Graduate Interns practicing in the city.

The method of receiving and distributing patients has been very much improved with the establishment of the new Reception Office, and patients may now be transferred from the ambulance or boat without direct exposure to the weather. All patients are admitted directly to this Reception Office, where their social history is taken, following which their clothing is re-

moved, and they are given a bath. Male patients are supplied with a clean pajama suit, socks, slippers, and wrapper; female patients are given a suit of underclothing, stockings, slippers, and hospital dress. They are then examined by the Examining Physician, and distributed to their respective Wards. Stretcher cases coming from the Ambulance Service are sent directly to the Wards, where a bath is given in bed by the Orderly or the Ward Maid.



Section-Cutting and Staining Room.

While in the Hospital the clothes of all patients are cleaned, mended, and pressed, folded and set aside on racks to await the patient's discharge. When ready to leave the Hospital, it is ascertained whether the patient has a home and if he is able to take care of himself. If in need of admission to a Convalescent Home, arrangements are made through the Social Service Bureau—a most valuable adjunct to the Hospital. Those lacking in clothing are provided for, and every reasonable effort made to help them to reach their destination. Every patient leaving the City Hospital to-day

does so in clean, neat, wholesome clothing, and with sufficient funds to reach friends or with a proper transfer to a Convalescent Home.

The Staff House, a three-story building for the resident interns, has been in successful working order for some time. It has twenty-eight separate rooms, four of which are double rooms, accommodating two doctors.

On the lower floor is situated the Dining Room, Library, and General Lounging Room, which is spacious and contains a pianola and pool table for use during recreation hours. The Superintendent's Cottage has also been opened, and affords a pleasant and healthful abode for the Superintendent of the Hospital and his family. A new kitchen, dining and service building of three stories is in active use. On the ground floor is a general storeroom, containing as well a butchers' shop and refrigerators. Here also has been installed an ammonia ice plant, with its system distributed throughout the building. The second floor is devoted mainly to dining rooms—two large rooms for the male and female helpers, aggregating 250 or 275 people, and three or four small dining quarters for the officers of the Hospital, numbering about sixty-five. On the third floor is situated the general kitchen, which affords food for the patients and employees; from this general kitchen about 1,100 persons are fed. A smaller kitchen, known as the "Diet Kitchen," is operated in conjunction with the general kitchen. Here all special trays and dishes ordered by the attending physicians are prepared by pupil nurses, under the immediate supervision of the dietician; it has all the most modern up-to-date machinery for cooking, including electric ice-cream freezers. The most recent addition is the laundry building—modern in every respect, and operated entirely by electricity.

Two new dormitories are in process of erection on the west side of the island.

MODERN DIAGNOSTIC METHODS IN SYPHILIS

BY

J. A. FORDYCE, M.D.

ATTENDING DERMATOLOGIST

MODERN DIAGNOSTIC METHODS IN SYPHILIS

By J. A. FORDYCE, M.D., ATTENDING DERMATOLOGIST

Read before the New York Academy of Medicine, April 2, 1914

Our present diagnostic methods of syphilis are those applied to the demonstration of the spirocheta pallida in a suspected lesion, the reaction of the blood to the Wassermann test, the findings in the spinal fluid, and the cutaneous or luetin reaction.

In endeavoring to properly estimate the modern diagnostic methods in the various stages of syphilis, we must not forget the importance of careful clinical training in recognizing the various stages of the disease. Clinical observation and laboratory methods supplement each other. Furthermore, the great majority of practitioners are not prepared to carry out the dark field or other necessary technique, and in many instances are situated away from large cities affording facilities for such examinations. A thorough knowledge of the multiform character of syphilitic manifestations, especially of the primary lesion, cannot therefore be overestimated, and unless the available methods are accurate it is better to be guided by clinical experience alone than misled by improperly made or interpreted laboratory tests. The value of these tests as confirmatory or negative evidence is a matter of daily demonstration to the physician who has them at his disposal, provided they are performed by carefully trained laboratory workers, but when carried out by the inexperienced or according to the methods calculated to be short cuts, they are positively misleading, entail a valuable loss of time to the patient, and only serve to bring them into disrepute.

It is no longer excusable to delay the treatment of syphilis until corroborative evidence in the form of a generalized eruption appears, as it has been demonstrated absolutely that the infection may be aborted by intensive treatment while it is still local and that we are more certain of a cure in a limited time in the stage of early dissemination. It is therefore incumbent upon us to employ our best diagnostic skill and every means at our command to establish a diagnosis at the earliest possible date. To this end the most valuable aid is the dark-field illumination for the demonstration of the spirocheta pallida in the lesion of an individual suspected of a syphilitic infection.

The spirocheta pallida is demonstrable by the dark field illumination, the India ink, the Giemsa and Levaditi stains and by inoculation and cultures. Owing to its simplicity and rapidity, the dark field is the method of choice. Clear serum only should be used, and with care and practice good

specimens are easily obtained. The organisms are usually fairly numerous when antiseptics have not been previously applied. If none are found, the procedure should be repeated for several days, and, if negative or doubtful, repeated Wassermann examinations should be made. Cutaneous chancres present little difficulty in the recognition of the spirocheta pallida, but in the mouth there is greater difficulty in differentiating it from the spirocheta dentium. In the latter, however, the spirals are not as regular as in the pallida. The India-ink method cannot compare in accuracy with the dark-field illumination, and the Giemsa smear and the Levaditi tissue stains are not practical for diagnostic purposes except in operation or autopsy material, nor are inoculation and cultural methods, their value being restricted to experimental work, owing to the length of time, five to ten weeks intervening between the time of inoculation and positive laboratory findings. The latter may prove of greater value in the future, as recently suggested by Nichols (*Journ. Exper. Med.*, Vol. XIX, No. 4, 1914), in prognosticating the type of infection from the cultural characteristics of the spirochetes obtained in a given infection. In a strain isolated from the nervous system he found that they had a thicker form, a shorter incubation period, producing in the rabbit hard lesions with necrotic centers and showing a tendency to generalize with lesions of the skin and eye following local inoculation of the testicle and scrotum. He is of the opinion that they possess a highly invasive power rather than a neurotropic action.

It frequently happens that after careful and painstaking search we fail to find the spirochetes owing to the use of antiseptic dressings or ointments which in many cases are applied by the patient before he presents himself for an opinion. In these cases we must depend upon clinical experience. In making a diagnosis of the initial sore, one must bear in mind its multi-form appearance; that chancres are often without induration or ulceration from the beginning or that such characteristics may disappear in a few days even without treatment. In the mixed forms, the chancroids sometimes remain nonindurated and constitutional symptoms are absent or overlooked until tertiaries or symptoms pointing to involvement of the nervous system appear. Urethral sores are sometimes unattended by local symptoms or these are attributed to an accompanying gonorrhœa. The primary lesion may manifest itself by the slightest abrasion or may simulate an outbreak of herpes, or there may be no discoverable chancre, the glandular enlargement being the only symptom. The hard edema sometimes seen on the foreskin means a spirochetel lymphangitis involving the entire circumference of the foreskin and producing a phimosis, the indurated edema of the labia being an analogous condition. Again, infection may take place without any local reaction, the organisms probably entering the lymphatic system direct. I have had a number of cases where by the most painstaking search it was not possible to find any evidence of an initial sore, but such cases were shown to be definitely syphilitic by secondary symptoms and a positive Wassermann. I have under observation at the present time a male

nurse who pricked himself with a needle used for taking blood for a Wassermann directly after he had withdrawn the blood from a very florid case of syphilis. There was absolutely no lesion at the site of the puncture, and although the patient watched for secondaries, nothing appeared for several months, when a few papules developed between his toes. His Wassermann reaction was strongly positive. Nonne also reports the case of a young physician who was pricked with a needle used on a syphilitic individual, in which there were no primary manifestations but secondaries and tabes three years later. Hutchinson likewise mentions several cases following a needle prick in which no local phenomena followed the accident but which in due time developed constitutional symptoms.

Insect contagion may in some cases be a means of direct inoculation without demonstrable chancre. A patient came to me several months ago with a fully developed secondary eruption and iritis. There was no history of an initial lesion and minute examination failed to reveal the remains of one anywhere. The patient had had, however, about two months previously, pubic pediculi, and it is not at all improbable that the infection was contracted in this way. He was an engineer returned from a three or four months' trip to the woods, during which time there had been no exposure. As to the manner in which the pediculi were acquired, he was not aware. Hutchinson narrates a similar case. He also believed that the association of syphilis with scabies was more than a coincidence. The diagnosis of extragenital lesions is frequently very difficult, not only because of their location in out-of-the-way places but because of their atypical appearance. An initial lesion under the nail, for instance, may be quite overlooked until secondaries make their appearance, as in the case of a dentist at present under my care, in whom a banal lesion was quite without the earmarks of lues. A dark-red spot then may be the only evidence of primary infection, or large phagedenic lesions bearing little semblance to a chancre may obscure the real nature of the trouble. Initial lesions of the lip, tongue, throat, eyelid, and other extragenital locations, especially when occurring in older people, are frequently mistaken for surgical or other infections. In the throat, differential diagnosis of the primary lesion from Vincent's angina not infrequently comes up. Sometimes patients are met with in whom there is an absence of palpable lymph node enlargement. This may be true not only of the area drained by the so-called satellite lymph nodes, but as is more usual there may be an absence of general adenopathy. In conceptional syphilis, of which we are as ignorant as ever of the manner in which infection takes place, we now know through the Wassermann reaction that not only the child but the mother, in the absence of all clinical manifestations, is definitely syphilitic. On account of the unusual way in which syphilitic infection may take place, it is extremely difficult to draw any definite conclusions as to the percentage of luetic infections in nervous and vascular troubles.

THE WASSERMANN REACTION

During the past four years I have had ample opportunity of comparing Wassermann reports from laboratories in various large and small cities of the country and find that a very large percentage are unreliable. The results are unreliable because negative reactions are reported in patients who have very positive manifestations of the disease and positive reports are received on patients who have never had any evidence and have never suffered from the disease in any way. In making a diagnosis of syphilis in doubtful cases, there is so much at stake in the question of treatment, the mental state of the patient, and his future health that we can only afford to take cognizance of tests which have proved to be trustworthy over a long period of time. We cannot condemn a man to a long period of treatment on the report of one Wassermann laboratory that has proved to be unreliable, nor can we condemn a method because of the conflicting reports when the technic and the laboratory are at fault. It is the consensus of the best authorities that the original Wassermann is still superior to all modifications which have been devised, and this should be taken into consideration when submitting a specimen for examination. The only departure from the original method permissible is in the matter of antigens, it having been demonstrated that the use of alcoholic extracts of guinea-pig, human and beef hearts fortified with cholesterin gives more delicate and more uniform results. This is particularly well illustrated in tertiary cases where the percentage of negative reactions with manifest symptoms has been markedly reduced. In performing accurate Wassermann work, no detail can afford to be slighted, and under no circumstances should the test be made without a preliminary titration of the hemolytic system for the day's work. It is very important that the complement be titrated, as guinea-pig serum varies greatly, and too large or too small a dose may give inexact results. Nor should one be content with the use of one antigen only as a slight variation in the strength of the extract or the method of mixing it may throw the test off, and for this reason a sufficient number of controls should be used.

What is the diagnostic value of the Wassermann reaction in the various stages of syphilis, and how are we to interpret it?

In the primary stage, its diagnostic value is overshadowed by the demonstration of spirochetes in the lesion. The greatest diversity is found in the statistics giving the percentage of positive reactions in the initial period of the disease, and this is obvious when we consider that they depend entirely on the length of time that the lesion has existed or rather that it has been observed by the patient, which is usually less than its actual age, and on difference in technic employed by various observers. The earliest date recorded in the literature is that by Craig, who found a positive reaction five days after the appearance of the sore. From his figures 27.6 per cent. give a positive reaction in the first week (Studies of Syphilis. Craig and

Nichols. Bull. No. 3, War Dept., June, 1913). On the other hand, Boas found that the sixth week and, as a rule, a much later date—the seventh or eighth week—was the more usual time (Die Wassermann'sche Reaction, 1914). From my own examinations the earliest reaction appeared at the end of two weeks, the majority after the fourth week. I have under observation a large number of patients treated within two weeks of the appearance of the chancre with positive microscopic findings and a negative Wassermann in whom the affection was aborted. Here the Wassermann is more important as a guide to the effect of treatment. To illustrate: A patient presented himself with a lesion of the penis of eight days' duration, slight inguinal adenopathy, spirochetal examination positive, Wassermann negative. Treatment was begun immediately, following the intensive plan of combined courses of salvarsan and mercury. He has never developed anything whatever as a clinical reminder, nor has his reaction become positive, so that to the best of our present knowledge a complete sterilization of his infection was attained.

It is frequently possible in early cases to follow a developing reaction; thus at the end of two weeks a partial inhibition of hemolysis manifests itself which does not hold, the cells going into solution the next few hours in the refrigerator. This incomplete fixation may continue for two weeks, at the end of which time inhibition will be complete and no hemolysis will take place over night. During this stage of development a strong reaction may be precipitated by an injection of salvarsan—in other words, a provocative reaction will be obtained. To cite the course of such cases, I was consulted by a young man with multiple erosions of the glans penis and foreskin of two weeks' duration in whom examination for spirochetes was futile owing to the application of various antiseptic solutions and ointments. His Wassermann at this time was $\pm$. This test was repeated several times, and the following week it became $+$. Two days later he received a salvarsan injection, after which the blood inhibited completely. In these cases with intensive treatment all clinical accidents are averted and the reaction is more readily permanently changed.

In the secondary stage of syphilis with the outbreak of cutaneous or mucous membrane manifestations, the reaction is positive in 100 per cent. of cases, and may continue so indefinitely in untreated cases. That it becomes attenuated and even negative after a lapse of years in a certain proportion of cases without treatment we know from findings in cases with tertiary clinical manifestations and negative serological findings and also in the case of women who have had repeated abortions or definitely syphilitic children and have themselves never suffered from any symptom of the infection. In such cases, although the Wassermann reaction may be negative or only weakly positive, we are justified in resorting to a provocative injection of salvarsan to develop a positive reaction.

The question of a differential diagnosis between the secondary eruption of syphilis and other skin affections which simulate it is often puzzling, even

to the trained dermatologist. Those which come under consideration are usually pityriasis rosea, drug rashes, forms of toxic erythema, and certain cases of acute generalized psoriasis. The presence of concomitant manifestations of the disease, such as adenopathy or mucous membrane involvement or the persistence of the primary lesion, enable us to make a diagnosis. Scaling syphilides sometimes cannot be differentiated objectively from psoriasis. I have noted, however, that in scaling syphilides with clearing in the center pigmentation is left which gives a clue to their nature, while in psoriasis pigmentation, if it occurs at all, is exceedingly rare. The final appeal in all these cases is the Wassermann reaction, which makes the diagnosis positive.

In the latent period of the disease the positive Wassermann is the only symptom of the infection, but scars of former lesions and the persistence of a pigmentary syphilide or leucoderma of the neck which may persist for two years or longer after disappearance of the secondary rash, is often a helpful indication of what has gone before. Statistics covering the positive reactions in the latent stage are of little value since we must take into account early or late latency, the amount of treatment, the length of time since cessation of treatment, and whether some of these may not be cured cases.

Many of the late latent cases show strongly positive reactions during active treatment with mercury and salvarsan, and it may be that we have fast or permanent fixation in these cases which we are unable to influence by repeated courses of treatment. Such reactions remain double plus. In cases that show tingeing, it means that treatment is beginning to be effective, and with further treatment we may be able to bring about complete hemolysis. In some, however, only incomplete hemolysis or an indeterminate plus-minus reaction is accomplished. This indicates the need of additional treatment if the amount preceding has been inadequate; if it has consisted of several courses, it is well to let the patient rest for several months, as it frequently happens that after intensive treatment the reaction gradually grows weaker over a period of months. If an interval of six months or longer has elapsed and it is still plus-minus, it is advisable to give a provocative salvarsan injection. Should the reaction become stronger then, we have an indication for continuance of treatment, but if it remains unchanged probably little significance can be attached to it, and it may be disregarded. In tertiary cases with lesions I have found that about 95 per cent. give positive reactions. Here again we are confronted with the questions of the amount and kind of treatment immediately preceding the test or at a more remote period. In the tertiary cases in which negative reactions were obtained, the patients had been under some form of treatment, so that the statistics in these cases have little value.

Especially in tertiary cases with gummatous lesions the Wassermann reaction is of great importance to differentiate from other conditions, as tumors. This corroborative testimony is conclusive in lesions situated on

the skin. When the mucous membranes or viscera are involved, we are dealing with a more complex problem, for the luetic may have a carcinoma independent of his syphilitic lesion or it may be engrafted on a leucoplakia of the tongue or cheek, several cases of the latter having come under my observation recently. One of them was a man, aged 37, who had contracted lues six years previously. He had a marked leucoplakia of the left cheek with a papilloma, which he had noticed but four months before, involvement of the lymph nodes of the neck, and a strongly positive Wassermann reaction. Histological examination showed a prickle cell epithelioma of very malignant type and a syphilitic lesion of the lymph nodes, the latter being very suggestive of the possible resting places of the spirochetes.

Leucoplakias usually excite the suspicion of a luetic process, but it is not infrequent to see them independent of the infection, the Wassermann remaining persistently negative even after a provocative salvarsan. In connection with the case just cited, I might mention that of another man, aged 48, who gave no history and who had never exhibited any evidence of lues. He had been an excessive smoker since his twelfth year. His left cheek was the site of a leucoplakia beginning about three-fourths of an inch from the oral commissure and continuous with a papilloma, which extended to opposite the last molar tooth, where it was ulcerated. The Wassermann reaction in this case was negative, and the microscopical examination confirmed the diagnosis of a prickle cell epithelioma.

Leucoplakias in non-smokers or in women where the influence of tobacco smoke cannot be evoked, in the absence of syphilis, are exceedingly rare. In these cases there must be a lowered resistance on the part of the mucosa to irritants which would not be effective except under conditions of this kind. I recently saw a woman, 47 years old, with an extensive leucoplakia on the dorsal surface of the tongue, chiefly on the left side, and on the mucous membrane of the cheek along the line of the closed teeth. There was nothing in the history of herself or husband pointing to lues, and both gave a negative reaction. Notwithstanding, she was given a provocative injection of salvarsan, but her reaction continued negative. There is then a small group of cases for which we have at present no etiological classification.

Other forms of cutaneous disease which sometimes simulate gummata very closely are tuberculosis, sporotrichosis, blastomycosis and persistent bromide eruptions. The latter may cause proliferation of the epidermis and vegetating growths which may progress serpigiously and persist for an indefinite time. Iodide eruptions also may produce erythematous and encrusted lesions which are difficult to differentiate from similar ones produced by the infection, as well as deep-seated infiltrations which are not unlike cutaneous gummata. They are, however, more acute in their development, are accompanied by more inflammatory phenomena, and disappear on the cessation of the drug.

THE PROVOCATIVE WASSERMANN REACTION

The reactivation of a negative Wassermann reaction by an injection of salvarsan, as first suggested by Gennerich and Milian, is now such a well-known biological phenomenon that it has found a place in the routine of diagnosis and treatment of syphilis.

To apply the test the patient is given an intravenous injection of 0.3 gm. of salvarsan and the blood taken on the succeeding days for a week. Different authorities report positive findings on various days from the first to the twentieth. In my own experience the majority of reactions have been obtained 24 hours after the injection. Occasionally in patients under treatment with a negative reaction it may be delayed until three or more injections have been given. An instructive case illustrating the value of the procedure is the following:

A., aet. 46. Luetic infection in 1886, indifferent treatment until 1910, when he began systematic courses of mercurial injections extending over two years and one salvarsan injection. In July, 1912, he discontinued treatment, and did not present himself again until December 6, 1913, complaining of subjective numbness of the right thigh and left arm. Neurological examination showed hyperactive knee jerks; right ankle jerk absent, the left diminished; the left pupil was fixed to light, poor to accommodation; the right sluggish; station, gait, and sensory apparatus were normal. His serum reaction was negative. A lumbar puncture made on January 2, 1914, gave eight cells, a faint excess of globulin and a negative Wassermann reaction. On January 5 he was given a provocative dose of 0.3 gm. salvarsan intravenously. His serum examined on the following day showed complete inhibition and has continued strongly positive to the present time during active salvarsan and mercury therapy. This case is probably one of beginning tabes of the root type. It would have been interesting to have performed another lumbar puncture after the provocative reaction was obtained in the blood, to ascertain if the same phenomenon occurred in the spinal fluid, but this the patient was disinclined to permit. Provocative reactions are obtained in the fluid two to ten days after the treatment, but with what regularity I am not prepared to say, owing to the difficulty of carrying out the procedure in private practice.

THE LUTIN REACTION

In 1911 Noguchi called attention to a cutaneous reaction which he obtained in inoculated rabbits and individuals infected with syphilis by the intradermic injection of a suspension of killed *spirocheta pallida*.

In the preparation of the emulsion a mixture of fluid and solid cultures of different strains of the spirochetes is rubbed together, the whole heated one hour at 60 degrees C. and 0.5 per cent. tricresol added. Of this suspension 0.03 to 0.05 c.c. is injected intradermically in the skin of the arm.

A positive reaction appears within four or five days, but may be delayed three or four weeks. Noguchi described three types of reaction in the positive cases—a papular, pustular, and torpid. In the negative cases a small erythematous area appears after twenty-four hours at the site of injection, unattended by pain or itching, subsiding in forty-eight hours, and leaving no induration. In some individuals slight papule formation may take place, but it is of no significance. The positive papular type reacts by the production of a large indurated papule in 24 to 48 hours situated on a red base. This increases during the next three to four days, when recession usually sets in with the disappearance of induration within a week. The pustular form begins the same as the preceding, central softening and pustulation taking place after three to four days. The pustule ruptures and becomes crusted, the margin remaining indurated. The crust falls off, induration recedes and healing takes place with very little scar formation.

In the torpid form the erythema which follows the injection fades within three to four days, then after ten or more days the process lights up and goes on to small pustule formation.

The injection is not accompanied by constitutional effects excepting slight rise of temperature, which may be present in positive cases for a day.

Positive reactions do not occur in the primary stage, and only in the secondary when treatment has been given. They are more frequently obtained in latent and tertiary stages, the reaction depending on specific sensitization, which only comes late in the disease. Nichols and Craig report (*Studies of Syphilis*. Bull. No. 3. War Dept. June, 1913) that there may be a provocative reaction similar to the Wassermann reaction after salvarsan. In a comparative study with the Wassermann reaction, they obtained in their secondary cases under active treatment 23 per cent. positive Wassermann and 38 per cent. positive luetin reactions. Their latent cases gave 25 per cent. positive Wassermann and 87.5 per cent. positive luetin. Combined latent and tertiary gave 53 per cent. positive Wassermann and 84 per cent. positive luetin. Noguchi's results (*Journ. Exper. Med.* Vol. XIV, 1911) were as follows: Manifest tertiary affections positive in 100 per cent.; latent in 94 per cent., and hereditary in 96 per cent. He, too, was under the impression that in the latent and tertiary cases the luetin reaction was more constant than the Wassermann. My experience with luetin as a diagnostic agent has not given as uniform results as the Wassermann reaction, but this may be partly explained by the fact that the original Wassermann was employed in my cases and also more sensitive antigens; namely, those fortified with cholesterin.

LUMBAR PUNCTURE

Another link was added to the diagnostic chain of lues in 1906 when Wassermann and Plaut discovered that the spinal fluid of paretics reacted positively to the Wassermann test. This was followed by a new line of

investigation on the neurological side of syphilis, which, however, was not placed on a practical basis until 1910, when Hauptmann and Hössli demonstrated that the amount of spinal fluid—0.2 c.c., the same used for the blood test—was inadequate to give the reaction in the vast majority of cases with involvement of the central nervous system. They showed that five times that quantity—or 1.0 c.c., with titration down to 0.2 c.c.—was entirely safe, gave quite constant findings, and enhanced the value of the test which up to this time was more frequently negative.

Spinal puncture in the hands of the properly trained is a simple procedure, not followed by untoward symptoms if all precautions are observed; but certain rules must be insisted upon. The patient should, to obviate such unpleasant symptoms as headache, nausea, vomiting, and giddiness, be kept in bed flat on his back for at least 24 hours. It has been said that patients with a normal fluid are more apt to suffer from headache than individuals with a definite pathological condition; but I have on several occasions met with severe headache lasting for several days in tabetics, as well as in normal individuals who have disregarded the caution to keep absolutely quiet.

In cases of cerebral tumor, the greatest care must be exercised, as here the withdrawal of the fluid is fraught with considerable danger. Nonne mentions several cases of sudden death.

The puncture should always be performed with the patient in the recumbent position, on the right or the left side, being a matter of personal preference. The back is bowed out by approximating the knees and chin. The skin is sterilized and anesthetized intradermically with a 2 per cent. novocain solution. A needle nine to ten cm. long and of 16-17 caliber is introduced between the third and fourth or fourth and fifth lumbar vertebrae, which is below the cord, and about eight to ten c.c. of fluid withdrawn.

The examination of the fluid consists of the estimation of pressure, the cell count, globulin test, and the Wassermann reaction.

The pressure of the fluid is estimated by attaching a graduated manometer tube to the puncture needle and reading the height to which the fluid rises. The pressure varies a good deal under normal conditions, and with movements, crying, etc. Only a definite increase, such as occurs with tumors or acute meningitis, is of diagnostic value.

The chambers for the cell count should be prepared at the bedside immediately after the fluid is withdrawn, as the cells settle very rapidly if it is left standing. The counts are made with the Thoma-Zeiss chamber and pipettes used for the ordinary leucocyte count. Two chambers should always be prepared. The diluting fluid, 10 per cent. acetic solution, is drawn to mark "I" on the pipette and the spinal fluid to mark "II". This is mixed, and after throwing away the fluid in the capillary part of the tube a drop is run on the counting stage, a cover glass is placed on the slide, and the cells on the ruled surface counted. As the chamber is one-tenth c.mm. in depth, the total count gives the number of cells in nine-tenths c.mm. of diluted

fluid. To get the number of cells in a cubic millimeter we multiply by 10 and divide by 8.

For the globulin test, Noguchi's butyric acid reaction is the most satisfactory. Two-tenths of a c.c. of spinal fluid is boiled over a flame with 0.5 c.c. of 10 per cent. butyric acid in normal salt solution; 0.1 c.c. of a 4 per cent. caustic soda solution is added, and the mixture heated again. The result is watched for ten minutes and then read at the end of one-half and twenty-four hours. The scale of reading runs from an opalescence which is negative to a coarse flocculent precipitate interpreted as a double-plus reaction.

The technic of the Wassermann reaction is the same as on the blood, except that a titration is performed on amounts varying from 1 c.c. to 0.05 c.c., or even lower.

Normal spinal fluid is a clear, slightly alkaline liquid, having a specific gravity of 1.006 to 1.008. It is a secretion of the choroid plexus and ventricle ependyma, having as its principal constituent sodium chloride with traces of carbonates and phosphates, dextrose, urea, and a faint trace of albumin. It is practically free from cellular contents, up to five cells per c.mm. being considered normal. The daily amount secreted averages 120 to 150 c.c., but in morbid conditions this is subject to considerable variation.

What is the significance of an increase in the constituents of the spinal fluid, and how are we to interpret it?

According to Nonne's teaching, 0 to 5 cells are normal; 6 to 10 are borderland cases, and over 10 constitute a pathological increase. A pleocytosis of the cerebro-spinal fluid then usually indicates meningeal involvement, but occasionally in other infections where the meninges take no part, as Landry's paralysis, pernicious anemia, and herpes zoster, a pleocytosis may be present. However, an increase in lymphocytes combined with other findings is strong presumptive evidence of syphilogenic disease.

A lymphocytosis is useful in differentiating cerebro-spinal syphilis and general paralysis from other conditions as neurasthenia and alcoholism, but it is more difficult to differentiate tabes, paresis, and cerebro-spinal syphilis from one another on the cytological count alone. This, in my experience, fluctuates within very wide limits, not only in paresis but in tabes, so that statements found in textbooks that five to twenty cells is a probable indication of paresis, twenty or over of tabes, and over 100 cerebro-spinal syphilis, have no value from a differential standpoint and show the fallacy of attaching too much importance to one feature alone. In paresis I have found a cell count as high as 280 and in tabes ranging from none to 247. It was formerly taught that lymphocytosis in tabes and general paralysis was uninfluenced by antiluetic medication and that this method was useful in diagnosing cases of pseudo-tabes and pseudo-general paralysis. This we now know to be untrue as with properly directed treatment the cell count is more readily influenced than either the globulin content or the Wassermann reaction. In a case of cerebro-spinal syphilis with 746 cells, the number

was reduced to 174 by one intraspinous injection of $1\frac{1}{2}$ mg. of salvarsan. In a case of tabes the cell count fell from 128 to 19 after an intraspinous injection of 1 mg. The cells are also markedly diminished by intravenous treatment alone; but this is much slower, as in a case with 150 cells the count dropped to 15 after five intravenous injections.

The globulin, or Phase I, reaction of Nonne and Apelt occurs exclusively in patients suffering from organic nervous disease, and is therefore of significance in differentiating between functional and central affections. While a moderate lymphocytosis may be met with in individuals who were once infected with syphilis and in whom the central nervous system has been spared, in Nonne's experience in 30 to 40 per cent. of cases, Phase I is never present unless there is coincident nervous trouble. Its presence will not assist in differentiating between specific and non-specific disease, but in general it may be said that the reaction is stronger when syphilis is the etiological factor. It is met with in infectious meningitis, in spinal cord tumors, and to a less extent and weaker in cerebral tumors. In other brain and cord affections it is weaker and not so constant. Recently I obtained the reaction in a case of leprosy.

It is estimated that globulin is present in 80 to 100 per cent. of cases of syphilogenic nervous disease, the variability in figures depending probably on antecedent treatment and remoteness from period of infection. Plaut reports 28 cases of cerebral lues, of which only 11 were positive, and adds that while a negative reaction does not militate against luetic disease, a positive reaction is to be taken as a serious symptom. Nonne is credited with saying that the endarteritic forms usually give a negative reaction. The cases which have come under my observation, diagnosed as cerebral and spinal endarteritis, have with only one exception reacted negatively to Phase I, unless an accompanying meningitis was present. This was a case of spinal endarteritis or transverse myelitis with four cells and a strongly positive globulin reaction and negative Wassermann. In the blood, the Wassermann was double plus.

It is generally agreed that the reaction is stronger and more constant in paresis than in other affections. There is usually a parallelism between Phase I and a pleocytosis, though this is not invariable, and the latter may be missing or very low when the former gives a strong reaction. With a high cell count, however, it is marked. In a case of syphilitic meningitis I saw at the City Hospital with a pleocytosis of about 2,000, the fluid coagulated solid.

The Wassermann reaction in the blood is estimated by Nonne and Hauptmann to be positive in 80 per cent. of cases of cerebral lues. The more recent the infection the more apt is it to be positive, while in old cases its strength depends on the activity of the process. In paresis the blood is positive in nearly 100 per cent. and this is used as a differential point from other forms of cerebral disease. In tabes the reaction is present, from Nonne's and Swift's figures, in 60 to 70 per cent. From my own about 66

per cent. gave a positive reaction. In the spinal fluid the reaction is positive in paresis in 100 per cent. of cases with amounts over 0.2 c.c., and the majority will titrate lower down than other types of cerebro-spinal lues. This point is considered a diagnostic feature by many authorities or as an indication that the type will eventuate in paresis or tabo-paresis.

In tabes the reaction in the fluid depends on the activity of the process, being strongly positive in the early cases with a cell count and positive globulin, and negative in those cases where purely degenerative changes are left.

It is not possible at the present time to give a characteristic serological picture for cerebro-spinal lues, tabes, and paresis. Bearing in mind that there are transitional or borderland forms, it is necessary to supplement laboratory findings with careful and minute clinical examinations. To obtain a proper conception of the serological findings in early tabes, repeated examination of the fluid should be made in all patients presenting symptoms pointing to involvement of the nervous system.

To summarize: The Wassermann reaction is one of the most important symptoms of syphilis, the interpretation of which can only come from experience—prolonged experience and careful comparison of laboratory results with clinical observation. There is great necessity for the standardization of the technic. All modifications introduced up to the present time have proven inferior to the original method, and cannot therefore supplant it.

A positive Wassermann is pathognomonic of a luetic infection with the following reservations: Partial or transient complement fixation is sometimes seen in leprosy, yaws, and cachectic states, but it is obvious that with the exception of leprosy, which is the only disease to present any perplexity in this climate, the clinical diagnosis would offer no difficulties. In the cases of leprosy which have come under my observation the reaction does not occur uniformly, some cases giving the reaction at one time and not at another, or negative and indeterminate with one antigen, while inhibition may be complete with another. An indeterminate or weakly positive reaction is of value as an indication for treatment but is of little importance for diagnosis unless as a concomitant of other symptoms when it may in many cases be brought out by a provocative injection of salvarsan.

In interpreting negative reactions, one must bear in mind that they may occur with limited tertiary affections, in old infections with the disease limited to the central nervous system, and in women with repeated abortions or syphilitic children in whom the infection is remote, and after treatment. Alcohol, taken prior to the test, will sometimes prevent the fixation of complement. Individuals who are addicted to its use should therefore abstain for several days before the blood is to be examined.

In the primary period of syphilis every effort should be made to establish a positive diagnosis. If the dark-field examination is negative and the clinical picture doubtful, repeated Wassermann examinations should be made, as this is usually positive before the outbreak of the secondary rash.

The Wassermann is positive in the secondary stage in 100 per cent. of cases, and is of value differentiating between early syphilitic eruptions and dermatological affections which simulate them.

In the latent stage it is a valuable guide to the effect of treatment. It is impossible at the moment to give the percentage of cases which will remain refractory to treatment and to say to what extent we are justified in continuing to treat such patients.

Untreated tertiary cases with manifest symptoms give a positive reaction in from 95 to 100 per cent. of cases. The test is here of diagnostic import in differentiating luetic affections from tumors or other surgical conditions and supplies the etiology in cases of repeated abortions where no history of syphilis is obtainable.

The provocative reaction is of great aid in cases with suspicious lesions and a negative reaction. It should be applied to all treated cases where the reaction has been negative for a period of a year or more to determine if a cure has taken place. If it becomes positive, there is an indication for renewal of treatment, and if it remains negative the provocative should be repeated within a year, following the policy of Gennerich.

All patients with symptoms pointing to involvement of the central nervous system should have a lumbar puncture, as well as the latent cases with persistent positive reactions, to determine if there is a latent process in the cerebro-spinal system. The puncture is of value not only in making a differential diagnosis between various neurological diseases, but as an index of the activity of a syphilitic process, and a control of the effect of treatment.

The findings in syphilis of the nervous system are as follows: In tabes the reaction in the blood is positive in 60 to 70 per cent. In the spinal fluid Phase I and lymphocytosis are usually marked in about 100 per cent. of well-developed cases with active manifestations. The Wassermann is positive with larger amounts of fluid and about 20 per cent. give a reaction with 0.2 c.c. or less. In the early forms of the root type the serological picture may be practically negative. Whether these cases will give positive findings later on, only further observation and time can tell. In the degenerative forms where all activity has subsided the results are negative.

In tabo-paresis and paresis the picture is about the same, *i.e.*, a positive reaction in the blood in about 100 per cent. of the cases; in the fluid a positive Phase I and a variable lymphocytosis with a positive Wassermann in large amounts in 100 per cent. and with 0.2 or less in about 95 per cent.

In cerebro-spinal lues the reaction in the blood is positive in 70 to 80 per cent., Phase I is usually positive, pleocytosis is variable, complement fixation is positive in larger amounts, a smaller percentage fixing with 0.2.

In cerebro-spinal arteritis the blood may or may not give a positive reaction. The findings in the fluid may all be negative excepting the globulin reaction unless there is an accompanying meningitis.

THE CIRCULATORY-RENAL COMPLEX

BY

HARLOW BROOKS, M.D.
ATTENDING PHYSICIAN

THE CIRCULATORY-RENAL COMPLEX

BY HARLOW BROOKS, M.D.,
Attending Physician

Notwithstanding the perhaps unfamiliar title, I do not propose to bring any new disease before you this evening, nor do I desire to rename an old one. My purpose is to collect under one name and as one disease picture a symptom complex founded on definite and reasonably constant pathological lesions and yet which is at times diagnosticated as arterio-sclerosis, although arterio-sclerosis may be but a minor and merely secondary lesion in the process; at times as cardiac disease, though the cardiac changes as a rule occur only rather late in the picture and most frequently of all as chronic interstitial nephritis, although the renal lesion is but a part of the picture and through the etiology, pathology, symptomatology and prognosis are quite different from those of a true nephritis.

The picture comprises a diffuse and simultaneous involvement of the arteries, usually also to lesser extent of the veins, and especially disease of the heart, the kidney, the liver, and of the brain. The involvement of the arterial system sooner or later pervades practically every ramification of the circulatory tree so that defective action may occur from the vessels of the brain to those of the lower extremities. Symptoms of renal disturbance, though perhaps of relatively minor appearance, ultimately develop; in the average case they dominate the picture in the middle stage which is commonly closed by signs of cardiac disease, although a considerable number of cases terminate in uremia.

The condition is of great importance, since it is certainly increasing in frequency and since it attacks by apparent preference those persons whose activities appear to be of greatest value to the body social. It is more common in my experience among men, though it occurs with probably equal frequency among those groups of women who are exposed to the same etiological factors which appear to largely determine the condition in men—that is, sex in itself has nothing to do with the development of the disease, which depends on circumstances of environment or of individual nature.

ETIOLOGY

As to the question of etiology, it is very probable that we shall to a large extent disagree among ourselves, yet this is one of the very most elemental

matters concerning the condition which must be cleared up before real progress in its management can take place. This divergence of opinion particularly applies concerning the rôle which alcoholism may play in the production of the complex. If we were discussing, for example, chronic interstitial nephritis, I think that there would be very little clinical disagreement as to the relation of this factor, although the actual experimental production of chronic interstitial nephritis by the feeding of alcohol is as yet decidedly in question. Alcoholism undoubtedly may play an important subsidiary rôle in the production of this form of disease, but it appears to be the conditions which are likely to accompany alcoholism rather than the drug itself which may cause the disease. A very considerable percentage of the cases in my experience have developed among those who use alcohol either very temperately, or not at all. Some of my most marked examples have developed among patients who have never used alcohol, and the disease appears to be relatively infrequent in Germany and Italy, where the use of alcohol is practically universal. Other poisons, such as mercury or lead, also seem to be unimportant in the production of these manifestations.

As regards tobacco, however, I believe that a quite different story will pertain, for in my experience the disease develops with a very considerable degree of frequency among smokers, and especially among those who use the weed to excess. There can be no doubt but that the abuse of tobacco makes the progress of developed cases more rapid nor that abstinence from its use, even among chronic habitués, causes improvement to follow. Syphilis is, I think, of rare primary importance, although of course it may be found frequently in conjunction.

There is one etiological factor which I believe will be found almost constant among the sufferers from this disease, and that is "stress of life." Physical excesses certainly do not produce the disease, whatever other deleterious effects they may have upon the heart, arteries, or kidneys. One rarely or ever sees this disease among those who live by manual labor unless this dominant factor of mental stress be also combined to a very definite grade. The complex is very frequent among our own ranks, among lawyers, and among the business men whose occupations entail, as most modern business seems to do, great worry and mental strain.

As subsidiary factors, chiefly secondary and largely matters of environment, come irregular or abnormal methods of life. One of the most important of these I believe to be irregular and insufficient rest or sleep. Either insufficient or over-sufficient food is a factor very commonly found in the history of these patients, and while there can be no doubt but that insufficient nourishment acts as a determining factor in some cases, over-feeding, particularly with the highly nitrogenous foods, is doubtless more frequent. In my experience a factor in this connection often overlooked, but apparently of primal importance, is concerning the matter in which the food is taken. When the meals are taken at irregular intervals and especially in irregular amounts, or when the food is rapidly poured in on the

digestive tract without giving the system an opportunity to properly care for or to metabolize it, bad results are very likely to follow. I leave you to speculate as you will concerning the mechanism by which these factors pertain. There are several very possible modes of action, but, be it as it may, the ultimate result appears to be undoubted. Other important etiological agents doubtless exist, but I shall not attempt to discuss them, for I believe that I have already mentioned the most important.

SYMPTOMATOLOGY

Among the very earliest of the symptoms are derangements of the gastrointestinal tract. The most frequent condition in this respect seems to be a gastric hyperacidity with corresponding flatulence, eructation, heartburn, and gastric distress, occurring with maximum intensity about one hour and a half after meals. Though more or less dependent on the character of food taken, that is, for example, worse when fats and sweets have been freely eaten, these symptoms also appear after the most simple of foods, especially when eaten rapidly or when the patient is laboring under excitement or stress. Oftentimes these patients will remark that they can attend banquets and formal dinners without the slightest distress, while the simplest of mid-day lunches causes very marked disturbance.

Constipation is very frequent, and a history of constipation alternating with attacks of dysentery or diarrhoea is common.

As a rule, there is little effect on the desire for food; and, as a general thing, the patients have a good appetite. Not uncommonly it may be voracious, even though the patient may fully realize that he is practically certain to suffer severely after eating. Depression of the appetite as a rule does not appear until late in the progress of the disease, and even then the desires may possess all the erratic characteristics of a chlorotic girl.

Indications of an unstable circulation are not slow to develop, though its recognition, as being definitely allied to any serious or general process, may not be apparent for some time. The earlier manifestations are rapid and irregular or intermittent action of the heart, especially an overly active cardiac response to nervous stimuli. Definite and independent heart lesions may not be demonstrable, and as a rule the muscle tone will be found good but the sounds of valve closure are sharper than normal, and from the heart sounds and activities a vascular hypertension may be suspected. Rapid flushing and blanching of the skin may appear, so that typical dermatographia may be demonstrable. Even slight thyroid enlargement or tenderness and a significant bulging of the eyeballs may be seen. More than once I have suspected the development of a Graves disease in these cases, and I am indeed yet far from prepared to deny but that hyperthyroidism may play some rôle in this disease process. Later, of course, signs of cardiac deficiency develop. Attacks of true angina pectoris may appear, but commonly the most serious indications are those of an insufficient heart muscle and of

impending dilation. Atrophy of the heart, usually of the brown variety, is found in some instances, hypertrophy in others.

The degree and type of arterial disease is extremely variable, and in this regard I radically differ from those who attribute this entire disease picture to arterio-sclerosis. Not at all infrequently well-marked cases appear entirely without arterial lesions of definite extensive grade either clinically or anatomically. As a rule, however, a greater or lesser degree of arterial disease does exist. This matter will be again discussed under the heading of pathological anatomy, but according to my studies the lesions are commonly of a form not correctly classed as arterio-sclerosis. Even though marked arterial disease may be demonstrable postmortem, little is often observable clinically, and the thickened, tortuous, and truly sclerosed vessels of senility or of true arterio-sclerosis will be usually sought for in vain, at least until the disease has progressed to such a point that secondary arterial changes are entirely to be expected. Janeway is evidently of the opinion that arterio-sclerosis may be the result, rather than the cause, of this complex (*Arch. Int. Med.*, 1913). I am rather inclined to agree with him. Hemorrhages are, however, frequent. They may be either capillary, venous, or arterial, and they may occur even relatively early in the course of the disease. Though they are naturally most frequent on the mucous surfaces or in the brain, they may occur in any distribution.

As a general rule, a more or less marked secondary anemia is present, and in some instances this may amount to a very definite hydremia with a tendency toward localized area of edema, urticaria, or even to real "dropsy" of the ankles, wrists, and face. There is nothing otherwise particularly characteristic about the blood findings in the disease, but in most instances you will find that this anemia is a very difficult one to relieve, even by the most approved methods of treatment.

High blood pressure, even a very high pressure with the systolic readings ranging well above 200 mm. Hg., is the rule in this disease in its fully developed phase. During the period of early development, the blood pressure will be found to be very erratic. At times it is high; then again after but a few moments perhaps it may be found normal or even sub-normal. This peculiarity is of some early diagnostic significance, for it will be noted that mental or physical excitement produces a rise disproportionately rapid and high, while depressive or exhaustive conditions may cause similarly a more than natural depression. When fully developed, however, the pressure is almost invariably constantly high until a stage of cardiac decompensation appears, in which sudden and serious drops in pressure are to be looked for and guarded against. This feature of the complex may be summarized as of three stages; that of erratic and irregular pressure, that of high pressure persistently maintained, and that in which sudden drops with circulatory and excretory decompensation occur.

The action of the kidney throughout is extremely puzzling, and is very likely to throw one off the track if he is inclined to rely for diagnosis on the

renal picture to a very large extent. I believe that the very first renal indication of the disease is irregularity of excretion. At times, and apparently without adequate explanation, an active diuresis may appear, and be followed perhaps by a period of more or less complete suppression, even with a urine picture which cannot in either case be said to be definitely abnormal. As a rule, the specific gravity of the urine is found much more consistently normal than sub-normal as would be expected in true chronic interstitial nephritis. The total solids excreted in the early or mean stages of the disease are, in so far as we are as yet able to ascertain, within the range of natural, though of course this depends very largely on the nature and amount of the foodstuffs taken. Casts, if present, are found in small numbers, and though hyaline predominate, a few granular ones are almost invariably found in those instances where any whatsoever are present. Blood rarely appears except where a more or less acute nephritis is engrafted upon the picture, an occurrence by no means rare. A striking feature of the urine is often apparent persistent normality, but systematic examinations will almost always show occasional traces of albumin. Later on when the condition has become fully established, the urine is typically that of a chronic interstitial nephritis. The main difference from the ordinary case of interstitial nephritis is that the urine for a long time appears to be practically natural, although from the general clinical picture a renal defect is almost certainly suspected. Another important fact of the same bearing is that life persists very much longer in these cases, if well managed, than is to be expected in true chronic interstitial nephritis. Dyspnoea is a very striking symptom, and often a very early one. I am not prepared to attempt its explanation.

As a rule, the liver presents few or no clinical symptoms, although anatomically it is commonly found to be the seat of a more or less diffuse fibrosis. The organ may be found either enlarged, normal, or atrophied.

Among the most striking manifestations of this disease picture are symptoms of central nervous origin which appear in most instances. In general, they may be said to be quite similar to those which develop so commonly in late chronic interstitial nephritis. In the early periods, the mental deviations are characterized, as it were, by their explosive character. These patients are usually irascible, cross, unreasonable, erratic and domineering, but such traits are likely to be contrasted with periods of optimism and jollity. These people are hard to get along with, and yet in the early periods no definite flaw can be demonstrated in the mentality, and as a rule they remain for a long time acute and clear in their cerebration.

Sooner or later, however, irascibility may become actual dementia, and unreasonableness true delusions of persecution, often with fixed ideas and a definite mental alienation as in many recognized types of legal insanity. This mental state should make the legal status of certain of the patients definitely one of question, for some of them are liable to commit in their disordered mental state all manner of injustices or crimes. A very brilliant and beloved member of our profession recently showed some of these

traits to a marked degree, and yet it was with some considerable difficulty that legal cognizance of his mental irresponsibility was established.

Another class of symptoms arising from diseases of the central nervous centers are those caused by hemorrhages. As a rule, these hemorrhages are not large, or the early ones may not be, and not infrequently they are followed by such complete absorption that they may be clinically considered as uremia, as of luetic origin, or due to vascular spasm. Where a patient lasts, however, sufficiently long, subsequent hemorrhages almost invariably appear, and these may be terminal in character, depending, of course, on their location or extent. As a rule, the small extravasations are followed by a considerable degree of recovery of function.

PATHOLOGICAL ANATOMY

The pathological anatomy of this condition is by no means a constant nor always a consistent entity. Cases differ very much in degree, not only in the relative grade of disease of the various structures but also in the exact microscopic type of the elemental changes. In the gastro-intestinal tract no characteristic alterations are demonstrable except that in most instances there is a more or less marked and constant congestion of the venules, and hence one must infer that the striking symptoms which point to disturbances of this tract are chiefly functional, metabolic, or chemical in nature, or are due to primary nervous disturbances.

In the heart a great variety of changes may be present. Apparently the most frequent one is replacement of the normal cardiac muscle by fibrous tissue, and it is notable that this fibrosis begins usually about the arterioles. Associated with these changes or entirely independent of them may be found a fatty degeneration or brown atrophy of the muscle. There is then nothing characteristic as to the pathological anatomy of the heart, although the heart disturbances appear to be clinically quite definite.

There is much diversion of opinion as to the most frequent lesion in the arteries; this fact in itself would apparently indicate that the vascular change is of very inconstant type or degree. In some cases extensive endarteritis is found, in others a fatty degeneration of the intima, subendothelia, and media is the dominant change. In my experience, the most frequent alteration consists of a periarterial fibrosis involving, at least most extensively, a very widely disseminated area, and it is seen in practically all the visceral trunks. This lesion may be best classified as an arterio-capillary fibrosis. I have found it absent, however, in quite typical instances of the disease, though I still believe it to be the most frequent characteristic of the vascular alterations. In some cases the vessel changes are so slight as to force one to the conclusion that the arterial lesions are really a result and not of real etiological character.

The kidney lesions microscopically are probably the most constant of any of those present in the condition. Essentially they consist of a fibrosis

usually showing definite evidences of activity. It is true that the kidney may be large and white, that it may be small, granular and either white or red in color, or it may be large, nodular, and red, thus covering a very wide field in so far as the gross picture is concerned; but in every instance studied by me the microscopic lesion has been practically constant. The degree of parenchymatous displacement or the type of its degeneration naturally ranges wide, but enough has been said, I think, to indicate that in so far as the kidney itself is concerned, the essential lesion is that of an active form of chronic interstitial nephritis. Indeed, I myself would be quite content to class these cases from their renal pathology as instances of chronic interstitial nephritis were it not that the etiology is so radically different in a high percentage of cases and the clinical picture and course quite characteristically divergent from that of chronic interstitial nephritis. At the same time, the involvement of other viscera and the prognosis as to duration are very different in the two conditions, facts which successful treatment must definitely recognize.

The anatomical change most commonly found in the central nervous tissues is an arterio-capillary fibrosis. Old or recent areas of hemorrhage, embolism or of infarction are of course to be expected, but cases exist in which very slight alterations indeed are demonstrable, notwithstanding that those same cases may have manifested characteristic mental symptoms. We must, however, admit that our knowledge of the finer ganglionic cell changes in the brain tissues is as yet very crude and that the nervous symptoms may be primarily caused by some toxic or metabolic process somewhat akin perhaps to those of uremia, whatever that may happen to be.

From this very brief consideration of the pathological anatomy of this symptom complex, it is very apparent that from the standpoint of the pathological anatomy we have certainly not shown this disease picture to be due to a true and definite morbid process. It may well be that later and more elaborate microscopic or chemical studies may demonstrate a universal explanatory lesion, but as yet the classification of these cases must rest entirely on the clinical signs, and my only attempt to-night is to indicate the clinical desirability of grouping these cases together for prognostic and therapeutic purposes.

THE DIAGNOSIS

Diagnosis is by no means difficult. The chief point lies in its differentiation from chronic interstitial nephritis. From this condition the early appearance of mental disturbances of an irritative character is among the most characteristic points. The preliminary development of circulatory instability, the erratic and finally high blood pressure, falling to or below normal only when decompensation develops, are all signs which can hardly be confused with those of a typical case of nephritis, when one has had the advantage of having the case under observation for a sufficiently long pe-

ried to admit of the study of these differential points. When the case is newly seen I do not think that an absolute differentiation should be made until after a sufficient period of observation, and for this purpose sphygmometric studies are indispensable.

THE TREATMENT

It is hardly possible to very extensively consider this or indeed any feature of this symptom complex in so short a period of time as is available to-night, yet I feel that my paper would lose its very object were I to neglect at least a brief discussion of the question of treatment—indeed, the object of my discourse is that I may point out to you that if begun in suitable time, treatment may yield very satisfactory results in this condition.

It is obvious that the first principle of treatment should be to check progress—that is, to promptly eliminate or correct as many of the etiological factors as possible. From what has been said in regard to etiology, it must then appear that the very first step should be to lessen worry and nervous tension. It has been pointed out that the harmful strain in most of these instances is of a mental type, and therefore it may well happen that the change of occupation from one entailing great mental stress or worry to one from this standpoint easier may be very beneficent. The popular cry of "Back to the Farm!" sometimes offers a possible solution for the brain worker. For persons of the leisure class, travel and sea or country life, particularly in the warmer climates and among quieting and peaceful surroundings, are of great curative value.

For most of us, however, and for many of our patients as well, such luxuries are not to be considered for economic reasons. A substitute treatment is usually, however, practical to a greater or lesser degree. If one should play all of the time for their health's sake but cannot, then perhaps the next best thing to do would be to play as much as possible. Outdoor recreations, especially such as are of a pacific character and which do not entail too much of the spirit of competition or strain, such, for example, as fishing, boating, moderate golf, and similar sports, may be recommended. Gardening, bee-keeping, chicken-raising, and the like may make it possible to combine business with pleasure to such a degree that life and comfort may be definitely prolonged. Where such diversions are impossible, quiet evenings or afternoon walks, reading, the theater, music, painting, modeling, and many other similar hobbies or pseudo-occupations, serve very much the same purpose. The effect of such measures, especially on the blood pressure and heart rate in the early forms of the disease, are wonderfully striking and by no means uncommonly progress is absolutely halted by such simple and agreeable methods.

Where circumstances permit, residence in a warm and even climate is very desirable, but life at a high altitude is undesirable unless the patients have been long accustomed to this altitude, in which instance they often do

better there than at a lower level. Climatic changes or alterations of altitude of an abrupt character are obviously bad. If circumstances do not permit of actual change of habitat, they may perhaps permit a patient to spend the more rigorous months of winter in the South, or allow a more or less prolonged vacation after the seasons of greatest stress. Hot or warm baths, either at spas or at home in the family bath tub, are very beneficial. A hot bath before retiring often acts as a more effective sedative or depressant of a hyperpressure than the most powerful of drugs. Even mild exercise may act in the same way not infrequently, especially if it be associated with pleasure.

Massage, electricity, passive and active movements may all be found beneficial. Their utility must and can only be judged by their results in each individual case and the best frequency of employment can be correctly estimated only from similar data.

Proper rest is always a very important factor, and at least eight or more hours out of the twenty-four should be spent in bed. As much time as possible should be spent in the open, and in my opinion sunshine is one of the most satisfactory agents possible for the depression of high pressure and for the promotion of increased elimination. It has much to do with the relative desirability of certain locations, and should be considered in the selection of rooms in the case of city dwellers.

The dietetic treatment is very important, but again it must be a matter of individual determination in nearly every instance rather than according to some predetermined rule. Naturally also the selection of diet lies somewhat on the nature of the work the patient is called upon to perform and on his previous habits in this regard.

In regard to alcohol, I believe that all the more concentrated forms should be entirely forbidden, although in some cases I do not object to whiskey, especially taken in the form of highballs—that is, well diluted. In some instances the very moderate employment of beer or of light wines I believe to do more good because of their nerve-vascular sedative effects than they do harm because of their pathologic results on the excretory avenues. Hot and irritating sauces or condiments must be restricted or wholly eliminated. I am convinced that ginger ale, for example, especially one largely flavored with capsicum, may not infrequently act as a more dangerous renal irritant than an equal amount of an alcoholic beverage. Salt must be used only in moderation; this especially applies to salt-cured foods because of the tendency exerted by sodium chloride in the retention of surplus fluid within the body. I am definitely persuaded that tobacco is bad in these cases. I advise that it be given up as a rule, but for those long accustomed to its temperate use and in whom it acts as a most welcome sedative and solace, its use in great moderation may be permitted. Since its harmful effects are chiefly evident from its action on the blood pressure, its effect may be quite satisfactorily determined by the simple estimation of the pressure before and after smoking.

Though nitrogenous foods as a general thing should be limited in amount, common sense must be especially exercised in this respect, for the diet must in every case be adjusted to the needs of each individual case, and the amount and character of meats in particular must always be made the subject of individualization. This is especially important, and it is often a very difficult problem of solution in those cases where gastric hyperacidity is present and in which, as a rule, foods of this questionable type are most readily tolerated by the stomach. Also, the problem of anemia must be considered and a diet sufficiently rich in iron must be provided. All things being equal, fresh green vegetables are very desirable; many of them, however, cannot be tolerated in instances where hyperacidity is present.

The amount and time of administration of water is another very important thing to be considered, since this also involves the desirability of soups and of the milk foods. As a rule, I think that the fluids need not be restricted except where the total urine output is less than the total fluid intake as fluid. Of course, this rough clinical method takes no account of the water in the solid foods, but it has been shown that that excreted by the bowel, skin, and respiration usually about balance this amount. It is commonly a very easy matter to determine when water is being withheld in the tissues, and then it is time to cut fluid intake down. Up to this point I commonly advise the rather liberal use of water, although of course hydremia is not to be encouraged in a process such as this in which vascular disease plays so dominant a rôle.

In regard to the use of such stimulants as tea and coffee, cocoa or chocolate, again I think that it is necessary to individualize. Coffee is by no means the awful poison indicated on the advertising pages from Battle Creek, and in some cases we can ask for no better diuretic or general stimulant than tea or coffee. In other instances, however, and particularly when they appear to upset gastro-intestinal digestion, they should be eliminated from the diet list. The same is equally true of cocoa and chocolate, which as beverages in themselves possess very little real food value. The question of sugar and fat in the food list depends absolutely upon their effect. It is by no means infrequent for these cases to develop a glycosuria; obviously such instances should take no sugar, but in other examples sugar as a food of high caloric value is very desirable indeed and is usually very acceptable.

The use of medicines in these cases resolves itself into drugs employed practically for the following purposes: (1) As sedatives; (2) to depress blood pressure; (3) to stimulate circulation; (4) to facilitate digestion and absorption; (5) to facilitate excretion; (6) drugs which may have a specific effect in checking the progress of the disease.

In this department of treatment, however, even more than in the others, we cannot fail to note that it is essentially symptomatic. I propose to consider the drug treatment very briefly, not because I underestimate its value, but because I believe that the average physician is well versed in the use of

drugs relative to the management of this condition and also because I believe that the foregoing measures are of greater necessity.

As to those drugs designed to produce sedation, a point which I believe to be of great import in the management of this condition, I know of none other which equals the bromides, and I confess a preference for the salts of sodium and of strontium. Chloral hydrate may be from time to time very logically employed, particularly when insomnia is a trying symptom and also when it is at the same time desirable temporarily to depress, at least to a certain grade, the tension in the arteries. Physical and psychical methods of treatment are, I believe, much more curative and lasting in their effects than the drug treatment designed for sedation, and I believe that sedatives are to be used symptomatically and for temporary effects chiefly, if not exclusively. Of course, it goes without saying that except in the terminal stages such agents as codeine, heroin, or opium should not be employed.

I do not think that depression of the blood pressure by the means of drugs is often desirable except in emergency as when hemorrhage is either present or threatened. As I have elsewhere stated (*Interstate Medical Journal*, 1914), I am of the opinion that except in rare instances depression of the pressure by the agency of drugs is not generally advisable in any chronic state of hypertension, nor is this point usually accomplished except when drugs are used to a gravely toxic degree. The occasional symptomatic use of the nitrites, of nitroglycerin and of chloral hydrate may, however, at times be strongly advised. I wish also to call attention here, as I have elsewhere, to the splendid effects of venesection in depressing blood pressure in suitable emergencies. There is one drug which doubtless sometimes acts beneficially as a hypertension depressant; it may be profitably long continued and its action is never harmful in this regard. I refer to the iodides, but please do not ask me how they reduce the pressure, although I am firmly convinced that they may do so.

Practically all of these cases sooner or later, unless previously terminated by some intercurrent state, suffer eventually from circulatory decompensation. Digitalis—and, next to it, strophanthus—are beyond doubt the drugs to use in these instances. Contrary to widespread, though erroneous, impression, digitalis will not raise the blood pressure except to restore it to a sufficient level when decompensation has been threatened, and its proper exhibition gives some of the most brilliant, though of course temporary, results which are achieved in these cases. A monograph might well be written concerning its desirability. Strychnia is as a rule contra-indicated, but must be used, as may also caffeine, camphor, adrenaline, pituitrin, and so on, in emergencies. For long-continued treatment, none of these drugs are desirable, except perhaps in much complicated cases.

Symptomatic treatment designed to facilitate digestion and absorption is obviously necessary and its general principles are so clear that I shall consume no time in their discussion.

Agents employed to facilitate excretion include none of the powerful

diaphoretics, such, for example, as pilocarpine, because defective secretion in this condition is so intimately blended with circulatory decompensation that this factor must be always considered with it. Diuretics may often be well used, however, and of course cathartics, among which I class as most frequently beneficial the salines and in emergency the cholagogues.

Under the heading of drugs which apparently have the effect of checking the pathological changes believed to be primary in the disease, the list must be headed by the iodides, and preferably by the iodide of potassium. It is most useful in cases entirely independent of lues, though just as efficient in specific instances. Mercury and salvarsan, cautiously given, act well in cases complicated by syphilitic infection, but I, and I think others, have also been much disappointed in the results following vigorous antisymphilitic treatment in even definite cases of this infection in the course of which this syndrome has appeared. Perhaps as well under this heading as elsewhere comes the use of iron for the relief of the anemia, of the antacids for the gastric hyperacidity, and so on.

In conclusion, while I am fully aware that from the standpoint of pathological anatomy this group of symptoms cannot be successfully grouped as a definite disease process, from the clinical picture and the progress I believe that we are justified in the assumption that for the purposes of treatment any other attitude is undesirable and that such a recognition is absolutely needful if we are to properly conserve the welfare of this important class of patients.

In regard to etiology, although I believe that I have in the main correctly stated the conditions under which the picture arises, I do not delude myself in the impression that I have fathomed its real etiology, which would seem to be very probably of obscure metabolic or chemical nature.

A CASE OF HODGKIN'S DISEASE INVOLVING THE
SPINAL CORD

BY

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A CASE OF HODGKIN'S DISEASE INVOLVING THE SPINAL CORD

BY EDMUND P. SHELBY, M.D.,
Attending Physician

N. V., a Greek peddler, 25 years old, was admitted May 12, 1912.

Family History: Father and mother living.

Personal History: Does not remember being sick.

Present Illness: Began in March, 1912, with fever, cough, and pain in chest. Was treated for "grip" by family physician. He recovered very slowly, and six weeks later was advised to go to Bellevue Hospital, where a mass was felt in the left hypochondrium. An operation was performed, and a gland the size of a hen's egg was removed by enucleation from the region of the pancreas. Other enlarged glands were left intact. The wound was packed and a drain left in. Sections of the gland were examined by Dr. Charles Norris and pronounced "probably Hodgkin's Disease."

Patient was removed to the City Hospital in May, 1912, where he came under the care of Dr. Brooks, and later under my care.

The Bellevue notes were obtained from the transfer slip which accompanied the patient.

On admission to the City Hospital, the physical examination showed a rather small but well-developed man. The skin and mucous membranes were pale. Superficial glands not enlarged. There is a vertical scar along the outer border of the left rectus muscle, at the center of which is a small opening discharging pus and feces. The abdominal muscles are ridged, but a mass can be felt on either side of the wound. The spleen is not palpable. Numerous fine râles are heard over the base of the left lung in the rear. There is an irregular fever of 99 to 102 degrees, with a pulse of 80 and respiration of 20. The blood count shows R. B. C., 3,000,000; W. B. C., 5,200; hemoglobin, 30. He complains of weakness, cough, and loss of appetite.

A bedside note one month later is as follows: "Patient gaining weight, fistula closed, tumor mass less prominent. T., 101 deg.; R., 20; P., 80."

Six weeks later another note says: "Patient gaining in weight, but still has slight cough at night. A few râles are heard over the bases of both lungs. Temperature, pulse, and respiration unchanged."

Two weeks later he was able to go to the yard in a rolling chair. The

improvement continued four weeks longer. The cough subsided and the blood count showed a slight increase in the number of red and white cells with an increase in hemoglobin from 30 to 50, but the temperature and pulse remained unchanged.

About the first of September he began to lose weight and the spleen became palpable, and from that time on the progress was steadily downward. The temperature gradually rose from 101 to 103 degrees, and the patient became nervous and irritable.

On the 30th of September he had a convulsion lasting several minutes.

October 10th herpes zoster developed in the right lumbar region, extending to the right thigh. Patient growing steadily weaker, extremities swollen.

October 13th had another convulsion, temperature subsiding.

October 27th had another convulsion, temperature now subnormal and pulse slow and weak, but patient eats ravenously and frequently has convulsive seizures while eating, involving chiefly the face muscles, which contract spasmodically and then relax for about a dozen times. The eyes are fixed, and turn downward and to the left for a minute or two, and then turn upward. After this there is a slow return to consciousness. Delirium with loud talking and gesticulation was present during the last two weeks.

Death occurred November 15th, eight months from the beginning of the illness. The absence of sore throat and of enlargement of superficial lymph nodes was noticeable.

Permission for an autopsy was not granted until three days after death. I am indebted to Dr. J. H. Larkin for the following autopsy notes:

Date of death: November 15th, 1912. Date of autopsy: November 18th, 1912.

Body extremely emaciated.

No external deformities. There is an old operation scar, six inches long, extending from the free border of the ribs down the outer edge of the left rectus muscle.

Pleura. Both lungs bound down in the pleural cavity by old adhesions. There is a small amount of free fluid in places.

Mediastinum. There is no involvement of the mediastinum.

Pericardium. Contains a small quantity of straw-colored fluid. Otherwise is negative.

Heart. Weighs 350 gm. The muscle is pale and flabby. The left ventricle is 3 cm. in thickness, right ventricle $1\frac{1}{2}$ cm., valves normal.

Left Lung. Is consolidated posteriorly, the consolidation being that of lobular pneumonia.

Right Lung. Is collapsed. Scattered throughout the lung are small, white, spheroidal nodules, some of which are a cm. in diameter.

Bronchi and pulmonary arteries normal.

Spleen. Weight, 650 gm.; measures $17 \times 11 \times 7$ cm. On transection it is studded over with a number of white, tumorlike nodules which seem to coalesce, giving a reticulated appearance to the spleen, in contrast with the

distended, congested lymph or splenic pulp tissue. The organ is firm and hard and cuts as if fibrous in places.

Suprarenal glands both normal.

Left Kidney. Weight, 150 gm.; markings fairly normal, capsule free. Scattered over the kidney are likewise indurated areas of a similar character to those in the spleen.

Right Kidney. Weight, 150 gm. Capsule is adherent. Organ is pale and presents numerous white, fibroid nodules.

Liver. Weight, 1,800 gm. On cross-section it shows a glassy appearance, is exceedingly dense and firm. Scattered over the cut surface are irregular, glassy-like nodules of the same appearance as those in the spleen. The organ is intensely congested.

Pancreas. Weight, 160 gm. On transection the organ shows a firm, indurated structure, but otherwise is normal.

The peripancreatic lymph nodes are extremely involved, some of which are 5 to 6 cm. in length. These nodules seem to coalesce with the lymph nodules along the aorta.

The entire aortic lymph chain shows enlargement and matting together of the nodes which invest the aorta from its bifurcation up to the transverse arch.

Spinal Cord. Examination of the spinal cord shows the outer side to be invested with a gelatinous type of tissue which is especially marked around the anterior and posterior nerve roots. The bony canal shows irregular hemorrhagic zones in close connection with the periosteum.

Brain. Permission not given to examine it.

Bone marrow of lower end of left femur shows numerous whitish nodules.

Anatomical diagnosis. Abdominal Hodgkin's of advanced chronic form, showing marked healing. General lymphatic involvement of liver, spleen, kidneys, lung, and bone marrow.

Histological examination of the tissues shows the lesion corresponds very closely with that described by Reed. Sections of the lymph nodes show a rather dense hyaline stroma with many large multinucleated cells, lymphoid cells, and a few eosinophiles. In many of the nodes the fibrosis was very marked. The nodules in the spleen, liver, kidneys, and lungs show the same picture seen in the lymph nodes.

The gelatinous tissue around the cord contains many blood vessels and hemorrhagic areas with an indefinite stroma, and beneath this pale area is a tissue made up of a rather marked fibrous stroma, which contains many large, irregular cells, some of which have multiple nuclei, lymphoid cells, and very many eosinophiles. This tissue is especially prominent around the nerve roots.

The picture corresponds so closely with that seen in the lymph nodes as to leave no doubt that the membranes of the cord are involved in the Hodgkin's process.

The pressure of the new-formed tissue upon the anterior and posterior nerve roots has caused granular degeneration and vacuolation of many of the nerve cells, but the cord itself does not show any abnormality.

I could find in the literature only one case of Hodgkin's Disease involving the spinal cord. In Warrington's case of "paraplegia resulting from actual erosion of the vertebræ and pressure upon the cord," it was found at the autopsy that a great mass of lymph nodes had eroded the bodies of the eighth to twelfth dorsal vertebræ and that the growth had pressed upon and extended downward along the dura mater of the cord.

In my case the bodies of the vertebræ had not been eroded, but the intervertebral disks seemed softened, and while the growth may have entered the canal through this channel, I think it more likely started in the lymphoid tissue around the nerve roots.

The lesion was entirely outside of the dura mater.

The chief diagnostic point in Hodgkin's Disease is the enlargement of one or more regional groups of lymph nodes, and as 76 per cent. of all cases show marked involvement of superficial lymph nodes, the diagnosis in the majority of cases is easily made. But when the disease is located in the abdominal or mediastinal nodes or in the spleen, the diagnosis can only be made by exclusion. In this case the diagnosis was in doubt even up to the time of autopsy. Some of my colleagues were inclined to consider the case as one of glandular tuberculosis, in spite of the provisional diagnosis of the excised node and notwithstanding a negative Van Perquet reaction. Lymphosarcoma was suggested, but the diminution in the size of the abdominal mass and the absence of enlarged superficial lymph nodes seemed to rule that out.

The prognosis in such cases is uniformly bad. The use of arsenic and of the X-rays may temporarily check the progress of the disease, but relapses occur sooner or later. A recent report by Billings and Rosenow suggests the use of an autogenous vaccine. These investigators have isolated from the lymph nodes of twelve cases of Hodgkin's Disease a "gram-staining, non-acid-fast, polymorphous, diphtheroid bacillus corresponding to the one found by Frankel and Much in smears and cultivated successfully by Bunting and Yates and by Negri and Mieremet."

The use of autogenous vaccines caused general improvement and diminution in size of the glands in each of their cases.

When these findings have been confirmed by others, we may be able to offer some hope to the victims of Hodgkin's Disease.

EXTRA-PERITONEAL RUPTURE OF THE BLADDER:
ITS SURGICAL MANAGEMENT

BY

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EXTRA-PERITONEAL RUPTURE OF THE BLADDER: ITS SURGICAL MANAGEMENT

BY EUGENE FULLER, M.D.,
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Extra-peritoneal rupture of the bladder, for a serious injury, has received little specialized study or attention, and when this can be said of a surgical condition of such magnitude it follows almost as a matter of course that its management has been, as a rule, inefficient and unsatisfactory. Some may criticize this statement on the ground that hospital statistics show that a large percentage of such injuries are at present discharged from hospital alive and with the record recovered. Still, if one will take the trouble to investigate these cases so discharged, it will be found that almost all of them have recovered only sufficiently to be discharged from hospital, and that most of them have been left invalided—the majority severely so.

Peritoneal rupture, on the contrary, has been carefully studied, and the surgery applied to it has been radical and eminently satisfactory. Severe violence directed against the suprapubic or perineal region may result in extra-peritoneal rupture. When violence is simultaneously exerted in both these directions, the bladder has small chance of escaping severe damage. The worst cases of extra-peritoneal rupture are those associated with fracture of the pelvis. In many such cases violence has been severe enough to cause general destruction and to make death inevitable. In these extreme instances surgery, at its best, may be ineffectual. It is in the cases uncomplicated by pelvic fracture that surgery has a chance to show its efficiency.

Extra-peritoneal rupture generally occurs in the region of the trigonum; less frequently above, under the space of Retzius; and occasionally at the side of the vesical neck. In most instances the injury consists of one tear, less frequently of two, and occasionally of even more. Multiple ruptures usually occur in connection with fracture of the pelvis, and some of them are in the nature of punctures caused by the sharp ends of the fractured bone. If a rupture is small and perhaps incomplete, the amount of urine extravasated may be correspondingly slight, the result being simply a circumscribed area of involvement. A case such as this, treated expectantly, can leave the patient with nothing more than a corresponding area of pericystic sclerosis. The symptoms arising from such sclerosis may be slight or severe, according to the extent and position of the lesion. In the great majority of cases, however, owing to the volume of the extravasation or to

a complicating secondary infection, expectant treatment invites a fatal termination. Following such injury, the extravasated fluid tends as it increases in amount—due to a continuance of vesical distention or to pus formation from secondary infection—to rapidly extend itself, so that after the extra-vesical space about the site of the rupture is filled, active burrowing follows. The directions which can be taken by such burrowings are various. One point, however, seems from my experience to be reasonably assured, and that is that the most dependent part of the extra-vesical space—in other words, the post-prostatic area in which the seminal vesicles lie—always becomes involved; primarily, when the extra-vesical rupture is, as is usual, at the base of the organ, and secondarily when it is elsewhere. This is a point of the utmost importance to be kept in mind, as will be seen in considering surgical treatment. Further burrowings point outside the pelvis. They may extend behind the peritoneum—sufficiently, perhaps, to involve the peri-nephritic region on one or both sides—or they may mount upward, filling the space of Retzius in the middle, or the inguinal regions to either side. The extension above the space of Retzius may result in a marked hypogastric tumefaction, liable to be mistaken for an over-distended bladder. Or, following extra-pelvic deviations of the pelvic fascia, the burrowing may invade the thigh, passing out of the pelvis under Poupart's ligament, generally externally to the internal iliac vessels; or it may reach the scrotum, following along and externally to the structures occupying the inguinal canal. The buttocks can be invaded, the paths being via the obturator or the great sacro-sciatic foramens. These last paths, however, are rarely taken, as they are narrow and devious. Extravasations can break through the pelvic fascia into the ischio-rectal space, the perineum, and elsewhere, but the assumption of such directions is not to be expected unless the trauma causing the bladder rent likewise breaks down other structures which would otherwise serve as natural barriers.

There are no strikingly characteristic clinical symptoms associated with extra-peritoneal vesical rupture, and it is on this account that oftentimes the injury remains undetected until inflammatory changes or burrowings in connection with the extravasation attract attention. Hematuria is present after the accident, perhaps to such an extent as to fill the bladder with clot; or it may be so slight as to cause no noticeable discoloration. Where the bleeding is so slight as not to cause the formation of clot, and where the laceration does not involve the vesical neck, there is not apt to be retention. There is always some pain on urination, and this may be severe. However, the pain associated with other parts which have been injured by the traumatism responsible also for the bladder injury may be so prominent as to divert attention, thus causing the discomfort connected with urination to escape observation. There is apt to be febrile disturbance, generally marked. As extravasation progressively develops, attendant symptoms arise, and it is these secondary symptoms, as stated, which frequently first lead the surgeon to a realization of existing conditions.

Diagnostic considerations as regards extra-peritoneal vesical rupture vary greatly in accordance with the time which has elapsed after the accident before surgical aid can be rendered. Where a surgeon sees a case promptly, the differential diagnosis between anuria, intra-peritoneal rupture, and extra-peritoneal rupture is most important. Where there has been considerable delay, time itself, for better or worse, has generally solved these questions, but left in all probability others. Where a case indicating bladder injury is seen promptly, the patient should be instructed to urinate. If he can do so naturally and the urine is free from blood, the chance is that there has been no vesical injury, and expectant treatment, as far as the bladder is concerned, is warranted. If he cannot urinate, a sterile catheter should be passed. If no urine and no blood is thereby revealed, the probability of anuria due to shock is justified. The abdomen, however, should be carefully watched, and if at the end of a brief interval on a second introduction of the catheter no urine should be found, a measured amount of sterile normal saline solution should be injected into the bladder. If the volume injected is caught in the return flow, well and good. If not, the abdomen should be promptly opened and an intra-peritoneal rent looked for—and closed, if found. If there be no intra-peritoneal injury, inspection should then be made to find if there exists any evidence intra-abdominally of extra-peritoneal vesical injury. To aid in this inspection, in case there should be no evidence, a catheter can be introduced and the distention of the bladder watched as fluid is injected. The abdominal wound should then be closed and the bladder opened suprapubically and drained. Such prompt drainage may anticipate any extra-peritoneal extravasation, allow the bladder to heal, and obviate a secondary operation such as will be necessary should there be any marked degree of extravasation. If the bladder contain blood clot, or if there be retention due to an involvement by the trauma of the bladder neck or deep urethra, then suprapubic cystotomy should be promptly performed. The bladder, on being opened, should be emptied and inspected, to locate injury. If there be then any indication of rupture involving the peritoneum, the abdomen should be opened as a secondary step in the primary operative procedure, and such a rent closed. In any event, suprapubic vesical drainage should be established. If suprapubic cystotomy discloses extra-vesical rupture and there is good reason to believe that the bladder has been opened before urinary extravasation to any degree has had time to take place, nothing further should be done. After such operation it may be advisable—even in case there be doubt regarding the question of extravasation—to delay operating to insure extra-vesical drainage until symptoms develop indicating the necessity for such further procedure.

It is the cases of extra-peritoneal vesical rupture that have not had the benefit of prompt surgery immediately following injury—a prophylaxis, as it were, against urinary extravasation—which especially deserve notice, as they represent the large majority. Before considering the question of how

these should be surgically treated, it may be well to remark on the inefficient treatment so generally employed, for it is from the study of bad results that good ones can be appreciated.

In most instances where extravasation has not burrowed sufficiently to produce external manifestations, it will be found that the bladder has been opened perineally or suprapubically, or perhaps in both directions. Where burrowing has been extensive enough to cause surface evidence or much localized pain, an incision into such part or direction is common, followed by the liberation of pent-up fluid and the maintenance of drainage. Although late cystotomy does little and perhaps no good, incision into the area involved by the extension of extravasation is very beneficial in that it probably saves life. Still, its fault is that it does not cure. This is because such incision only partially drains the area. Another error into which those cutting into such burrowings may fall is observed in connection with cases where the burrowings have been sufficiently extensive to point outside the pelvis, far from the original source. When such a pus cavity is opened, the false inference may be drawn that a near-by part is the source of suppuration and consequently requires surgical removal. As an illustration: The head of the femur has been resected, the suppurative process having in reality burrowed out of the pelvis by way of the great sacro-sciatic foramen; or a piece of the sacrum has been chiseled off, on the supposition that disease there was the original cause.

In a paper entitled "Drainage of Extra-Vesical and Extra-Peritoneal Suppurations of the Male Pelvis" (Journal of the American Medical Association, October 25, 1902), which represented my early surgical experience, I made the following remarks:

"The surgical management of suppuration in this part or elsewhere lies in the establishment of free drainage. In order that drainage be free, it must be dependent. As the pelvic cavity is deep, the surgical problem of effecting dependent drainage arises. In each individual case there are two surgical principles to be determined in connection with the establishment of drainage: First, the location of the dependent pocket of suppuration; and, second, the dependent dissection to reach that pocket. No dependent dissection should be attempted until the pocket has been located.

"The second step in the surgical procedure is the dependent dissection. This dissection should open a perineal path and expose the lymph space between the prostatic capsule and the rectum, the urethra being left intact. In making this dissection, the dorsal position and the Zuckerkandl incision can be employed; but what is far superior is the knee-breast position and the incision which I devised to expose the seminal vesicles and the prostate, a description of which has been published in the Journal of the American Medical Association, May 4, 1901. . . ."

This early opinion, though thoroughly sound, I have since seen fit to modify as regards procedure, in that I now make the second step the first, while the original first step becomes the second. This change simplifies

matters and avoids exposing the patient to what often resulted in a needless degree of dissection in locating the pocket in connection with the original first step of the operation. As all these extravasations tend to speedily reach the most dependent extra-vesical space, that space should first be opened. It will then be found a much simpler matter to trace with the finger or probe the track of the burrowing from its dependent position to its limit of extension, than was the vice versa original procedure. When the limit of extension is located, an external incision can be made down onto the end of a probe and drainage tubes introduced. It may be that burrowings have taken place in more than one direction, in which event more than

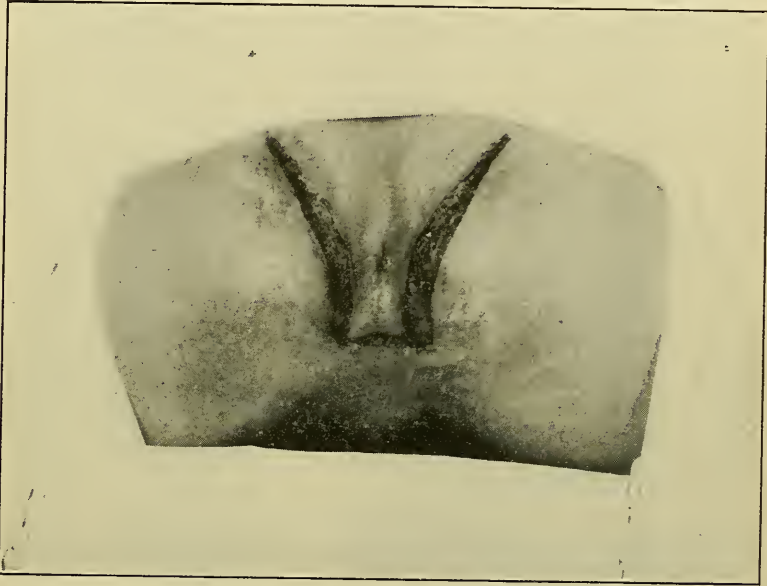


FIG. I.—Line of Incision (Fuller).

one external connecting incision will be necessary. Drainage tubes should be serrated and of sufficient length to pass in at the external connecting incision and out at the entrance to the dependent incision.

The accompanying photographs illustrate the performance of the dependent dissection. As the seminal vesicles in these cases are imbedded by infiltrate, it is wise to open them just as would be done in the performance of a regular seminal vesiculotomy.

Figure I represents the patient in the knee-breast position, ready for operation, and shows the line of incision. In this photograph, although the depth of the incision is still superficial, the marked tendency to retraction of the tissues is clearly evident. This tendency to retraction, intensified by the position of the patient, is an element of great aid to the operator. The

converging cuts on either side of the rectum are made long so that the rectum can freely retract as the perineal dissection liberates it from its natural anchorage.

Figure II indicates the method of perineal dissection. Note the marked retraction of the rectal structures, the lower rectal flap having already retracted fully two inches. This retraction is sufficient to expose the prostate on lifting the rectal flap, making that organ a superficial rather than a



FIG. II.—Perineal Dissection (Fuller).

deep-lying structure. In making this dissection, the forefinger of the operator's left hand is maintained in the rectum to serve as a guide while the dissection progresses. While so operating, the side rather than the blade of the scalpel is used, so as not to cut into the muscular structure of the perineum, the path of dissection lying just outside the muscular perineal body and inside the perineal fascia. By so doing, and by starting the transverse flap well below the margin of the anus, the rectal sphincter and the blood-vessels encircling that organ escape injury. In fact, so little injury is done to blood-vessels that it is exceptional when it is found necessary to

ligate a single vessel through the performance of the entire operation. As the dissection proceeds around the perineal body, that structure tends of itself to retract downward. The fascia propria of the pelvis, with its median raphe, is then exposed, and when this is cut through the bursal space separating the rectum from the prostatic body is entered.

Figure III shows the next step in the operation, the separation of the



FIG. III.—Separation from the Rectal Wall.

rectal wall from the underlying prostate, the seminal vesicles, and the bladder floor. After the fascia propria of the pelvis has been divided, the scalpel is put aside and the right forefinger of the operator is introduced into the bursal space just mentioned, and the process of separating the rectal wall from the prostate and seminal vesicles by digital manipulation is undertaken. In accomplishing this, the right finger-tip should be bent down to guard against perforation of the rectal wall, while the left forefinger is still maintained in the bowel as an aid in the same direction, for should the right finger impinge too closely on the bowel wall in accomplish-

ing its separation, the left finger gives the operator immediate warning. In the performance of this digital separation it may be that fibrous bands are encountered, too dense for separation by the finger-tip. If such is the case, the scalpel guided by a director can be introduced to cut the bands, after which the digital separation is resumed.

Figure IV illustrates the putting in place of the grooved director under the guidance of the operator's right forefinger. After the rectal wall has



FIG. IV.—Placing the Grooved Director (Fuller).

been detached from the underlying seminal vesicles, the operator places his right forefinger-tip on the apex of a seminal vesicle (in the photograph it is the apex of the right organ) and maintains the finger-tip in that position, while with the left hand the grooved director is passed under and along the right forefinger till its point reaches the apex of the seminal vesicle under the right finger-tip. In this position the director is firmly held while the right forefinger is withdrawn.

Figure V shows the next step in the operation, which is the passing of the knife along the groove of the director. After the knife reaches the apex

of the seminal vesicle at the point where the end of the grooved director is fixed, its shaft is lowered so that the belly of the blade lies against the seminal vesicle, and a cut of an inch and a quarter to an inch and a half is made longitudinally into the seminal vesicle so as to lay that organ open its entire length. Oftentimes the first cut may not be deep enough to accomplish its purpose. If a digital examination shows the cut to be incomplete, the director should be replaced and a second cut made in the line of the



FIG. V.—Passing the Knife (Fuller).

first. Sometimes, especially in sclerous cases, three or more cuttings may be required before a thorough opening of the seminal vesicle has been accomplished. Should it be desired to open the floor of the bladder for drainage, the end of the director should be placed against the wall at a point midway between the seminal vesicles and far enough back to be in a line with their apices. The knife should then be slipped along the groove and the bladder wall perforated, after which the director should be immediately slipped into the bladder and maintained there to guide the introduction of a drainage tube.

Figure VI shows the method of packing the cavities of the seminal vesicles and the deep limits of the dissection. Two strips of gauze are employed, one for each side. Here the packing is being introduced into the right opened seminal vesicle, the right forefinger of the operator being maintained in the opening to crowd in and arrange the placing of the gauze as it is being pushed in by the operator's left hand, aided by a blunt-ended clamp. In placing the packing, a space should be left between it and the rectal wall for the drainage tubes.



FIG. VI.—Placing the Packing (Fuller).

Figure VII shows the two drainage tubes in place above the packing, the lower portion of the flap of the dissection having been fastened in place by lateral sutures.

Figure VIII shows the completed operation previous to the placing of the dressing. The two ends of the gauze packing are left protruding, to facilitate removal. Safety pins have been passed through the tube ends, and to these have been attached strips of adhesive plaster, so as to guard against a premature slipping out of the tubes.

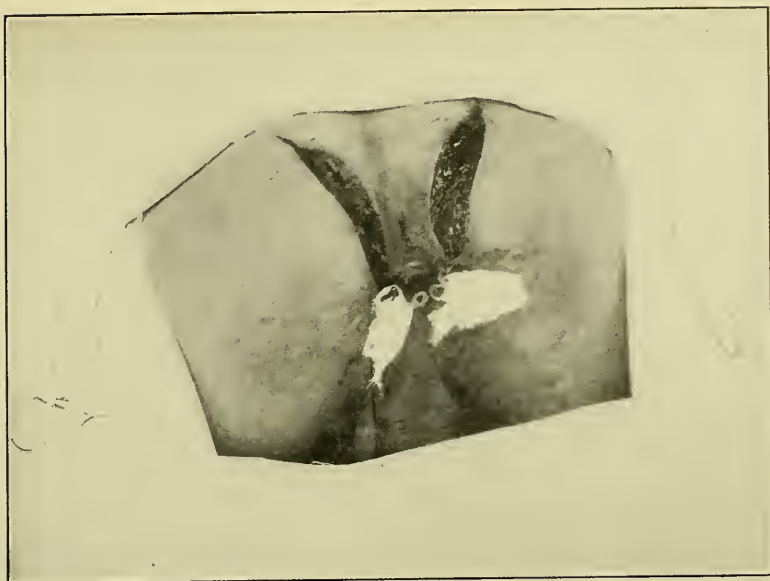


FIG. VII.—Drainage Tubes and Packing (Fuller).

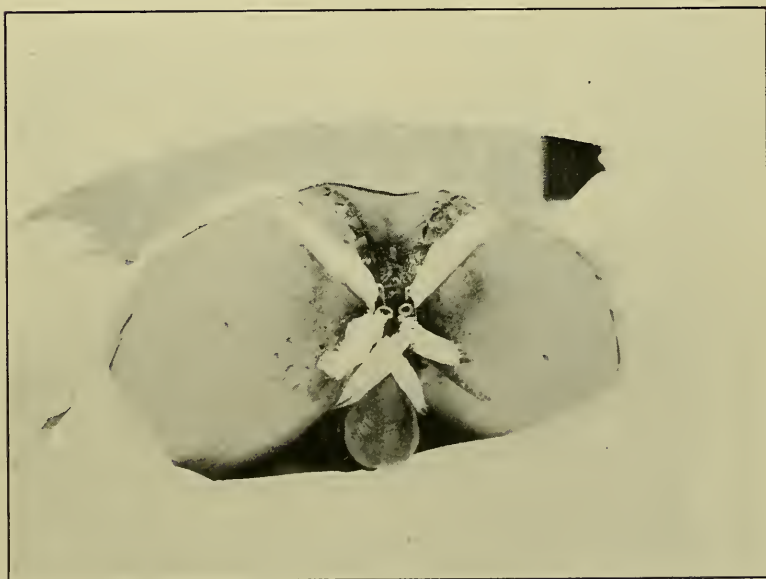


FIG. VIII.—The Completed Operation (Fuller).

The following clinical reports serve to illustrate the surgical procedure advocated in cases coming under observation long after the original injury:

Case I, 23 years old, on entering the City Hospital, could not walk without support, nor could he void urine. All the urine leaked away through a perineal sinus. There was constant and severe pain in the region of the pelvis.

Several months before, while working in a mine, his pelvis had been caught between two cars. Retention followed the pelvic injury. He was sent to a local hospital, where a perineal opening was made, and it was out of that opening that the urine was passing when he entered the City Hospital.

An X-ray picture showed that there had been a fracture of the pelvis. The injury had also caused thorough disorganization of the deep urethra, so that complete occlusion had resulted. Rectal examination showed marked tumefaction of the post-prostatic tissues, and any pressure there caused extreme pain. The patient was put in the knee-breast position and the seminal vesiculotomy dissection was made. Before this dissection had extended beyond the prostate, the anterior limit of the dependent area of extra-peritoneal extravasation was opened, and when completed a large chamber was exposed occupying the entire post-prostatic space. Communicating with this chamber was an opening through the trigonum sufficiently large to admit the forefinger. To the right of the vesical neck, the chamber extended upward into the space of Retzius. A suprapubic counter opening was consequently made, and a connecting chamber occupying the space of Retzius was thereby exposed, and another vesical rent found connecting the bladder cavity directly with the space of Retzius. From the space of Retzius, the burrowing had extended to the left under Poupart's ligament and into the upper thigh. An opening was consequently made into the upper thigh so that a drainage tube from the suprapubic opening could be there passed out. A suprapubic drainage tube was also passed through the space of Retzius to the right of the vesical neck, through the post-prostatic space, and out at the base of the perineal dissection. Another good-sized tube was passed along the perineal incision and into the bladder through the rent in the trigonum. This last was for vesical drainage. The operation was followed by marked relief.

Two months after this first operation, the deep urethra was resected and made pervious. The patient now—four months after the first operation—is about, using a cane, in comparative comfort. The suprapubic opening into the space of Retzius has not as yet been allowed to close, so as to afford a vent in case of persisting suppuration. The flow of urine, likewise, has not yet been diverted from the perineum to the urethra. The perineal opening can, however, be closed by a slight plastic operation whenever it may be deemed advisable.

Case II. A man, 31 years of age, entered the City Hospital with a history that two years before a log had fallen upon him, causing rupture of

the bladder, together with laceration of the deep urethra. Suprapubic cystotomy had, at the time, been performed. Eight months later, a perineal section had been performed to rid the patient of traumatic stricture formation and to avert threatened retention. His chief complaint on entering the City Hospital was impotency, which had been complete ever since the accident. On this account, his wife had left him. There was also marked pain in the sacral region after much exertion, together with loss of force to the stream on urination. Rectal feel showed marked infiltration and tumefaction of the post-prostate tissues, digital pressure causing severe pain. There was still a marked traumatic stricture involving the deep urethra. Before the accident, the patient had been normal and vigorous sexually, and there was no antecedent history of gonorrhœa.

Being of the opinion that the post-prostatic tumefaction was due to urinary extravasation, the patient was put in the knee-breast position, and the seminal vesiculotomy dissection employed. A burrowing extravasation cavity, in which lay both seminal vesicles, was opened. This cavity was found to communicate with the bladder by way of a rupture through the middle of the trigonum. This rupture was large enough to admit the tip of the forefinger. The extravasation had not burrowed beyond the confines of the post-prostatic space: The involvement of the seminal vesicles evidently accounted for the impotence. Both these organs were opened, as in an ordinary seminal vesiculotomy, and their cavities, as well as that of the extravasated area, packed. A perineal tube for urine drainage was passed into the bladder through the opening caused by the rupture.

Two months after this operation, the deep urethral stricture was resected. This patient is now completely well, has regained his sexual stamina, and is at work as a laborer.

Case III. A man, 40 years old, came to City Hospital with the history that seventeen years before he had fallen fifteen feet, striking his sacrum against a wheel. Before the accident he had been strong, and up to that time he had never had sexual intercourse. After the accident, he was in bed for a long time, and had great difficulty with urination, together with pain—but as the accident had occurred seventeen years before it was impossible to obtain exact clinical data. Four years after the accident, he married and had one child. He had never been well since the accident, owing to severe sacral and suprapubic pains, increased on exertion. These pains were often accompanied by chills or chilly sensations. The sexual function had never been strong since marriage, and for many years he had not indulged in coitus at all, owing to the fact that the act served to exacerbate his pains and chills. At present he had no complaint regarding urination, and the urine was normal. He had a great dread of urethral instrumentation, as such treatment caused him much after-suffering from an exacerbation of his symptoms. Neurasthenic symptoms were marked. In fact, neurasthenia had been the usual diagnosis given to account for the symptoms. Rectal feel showed the post-prostatic space filled with scar

tissue which involved both seminal vesicles. Digital pressure against this region provoked great pain and could not be tolerated. There was no stricture. The cystoscope showed little except that in the region of the trigonum the blood-vessels were enlarged and irregular. There were also in that area patches of scar tissue.

The patient was put in the knee-breast position and the seminal vesicotomy dissection employed. In this case, no actual extravasation cavity was found, probably owing to the fact that the accident had been so long ago; but dense cicatrix was cut through, the floor of the bladder liberated, and also the seminal vesicles, after which both these organs were opened. The space was packed and drained.

Complete recovery, with relief of all symptoms, followed.

REPORT OF A CASE ILLUSTRATING THE VALUE OF
METHODS USED FOR TESTING THE PERME-
ABILITY OF THE KIDNEY IN THE
GENITO-URINARY SERVICE OF
THE CITY HOSPITAL

BY

ROBERT HOLMES GREEN, A.M., M.D.,
ATTENDING SURGEON, GENITO-URINARY DIVISION

REPORT OF A CASE ILLUSTRATING THE VALUE OF METHODS USED FOR TESTING THE PERMEABILITY OF THE KIDNEY IN THE GENITO-URINARY SERVICE OF THE CITY HOSPITAL

BY ROBERT HOLMES GREEN, A.M., M.D.,
Attending Surgeon, Genito-Urinary Division

For several years past studies have been made in the wards of the above Hospital by the writer in estimating the value of the various methods advocated from time to time for testing kidney permeability. As is well known, one method will be popular and warmly, not to say enthusiastically, advocated by many members of the profession, then its popularity will wane and another one will replace it in popular estimation. It would simplify our work in many directions if the true scientific value and the popular accord given to various measures coincided, but as we all know very frequently such is not the case. Generally speaking, during the past four years my investigations have covered three periods.

A period several years ago in which I tested the comparative values of methyl blue and phloridzin. Then a period about four years ago in which I compared in a series of cases the value of pheno-sulpho-phthalein with phloridzin, and during the past winter in which I conducted examinations on a series of cases to show the comparative results as regards accuracy of pheno-sulpho-phthalein, phloridzin, and the estimation of the nitrogen in the blood compared with the nitrogen in the urine. This latter procedure has only been rendered possible since the laboratory has been put under the able management of Dr. Larkin, who is so ably assisted by the careful analytical work performed by the chemist, Mr. Lewis Pine, who had charge of the carrying out of the examinations for estimating the amount of nitrogen.

A more detailed report of these investigations is being reserved for a future report. Somewhat roughly speaking all of these latter tests were found to be good. As illustrating their accuracy the report is given below of a case in which the clinical symptoms pointed strongly to a chronic disease of the kidneys, heart, or both, but in which the findings by these tests indicating the kidneys to be sound, were confirmed by the autopsy.

Patient McNair, age 84, entered the hospital with an acute cystitis. Frequent careful examinations failed to reveal any obstruction anywhere in the urinary channel. Cystoscopic examination apparently showed arteriosclerotic changes in wall of bladder, which were not confirmed by an

RESULTS OF THE PHENO-SULPHO-PHTHALEIN TEST
IN KIDNEY ELIMINATION

BY

ORRIN S. WIGHTMAN, B.A., M.D..

RESULTS OF THE PHENO-SULPHO-PHTHALEIN TEST IN KIDNEY ELIMINATION

BY ORRIN S. WIGHTMAN, B.A., M.D.,
Attending Physician

For several years past the medical profession has been interested in securing some method which might facilitate a method of estimating the work done by the kidneys. Of those at present under consideration, much is being said about pheno-sulpho-phthalein, and with a desire of finding out its merit and clinical application, a series of cases were conducted at the City Hospital.

The study was conducted with a desire to get all the good the test had to offer; to find its limitations and advantages, and to see frankly what could be learned that could not be secured from other sources of examination. In treating this subject from a clinical standpoint, we took the broader ground of making a careful study of the patient's true condition and using the test as a check.

In the list of cases observed we were first impressed with the clinical picture of cardio-renal conditions in which the law of compensation first depended upon heart and then upon kidney elimination for even the semblance of body balance.

We felt after a while that the cardio-renal system was a very intangible something, which went further than merely recording the output of urine, representing also an unused reserve both of heart and kidney tissue which might, under proper conditions, be restored to a semblance of usefulness.

The technic of the test was of major importance, not only as a comparison with other investigators, but also to assure a uniform result in the cases observed. For this reason, two of my house staff conducted them, taking the average reading as observed by both. Our routine method was to catheterize the patient; give him a glass of water, and within ten minutes inject deeply into the quadratus muscle 1 c.c. or one ampule of the pheno-sulpho-phthalein. In some instances a catheter was left in situ with a pan containing a 10 per cent. solution of caustic soda to observe the rapidity and first indication of dye in the urine. Other cases merely voided the urine.

We used a stronger solution of urine for reading on the scale so that we might get a deeper color for comparison, and divided this color indication when totaling color. In other words, when as per the regular method it

states that the urine collected should be alkalined and then diluted to 500 c.c., we usually diluted to 250 c.c. and obtained a strength of color in the urine which was more easily read.

We had some difficulty where there was a heavy deposit of uric acid, urates, or bile pigments in getting a urine for comparison which was of the clear pink tone of the standard solution. The urine was apt to be tinged slightly brown. This especially when the amount of color voided in the urine was small. A north skylight was used in reading.

In order to get as complete a picture as possible, a regular History Record was printed as follows:

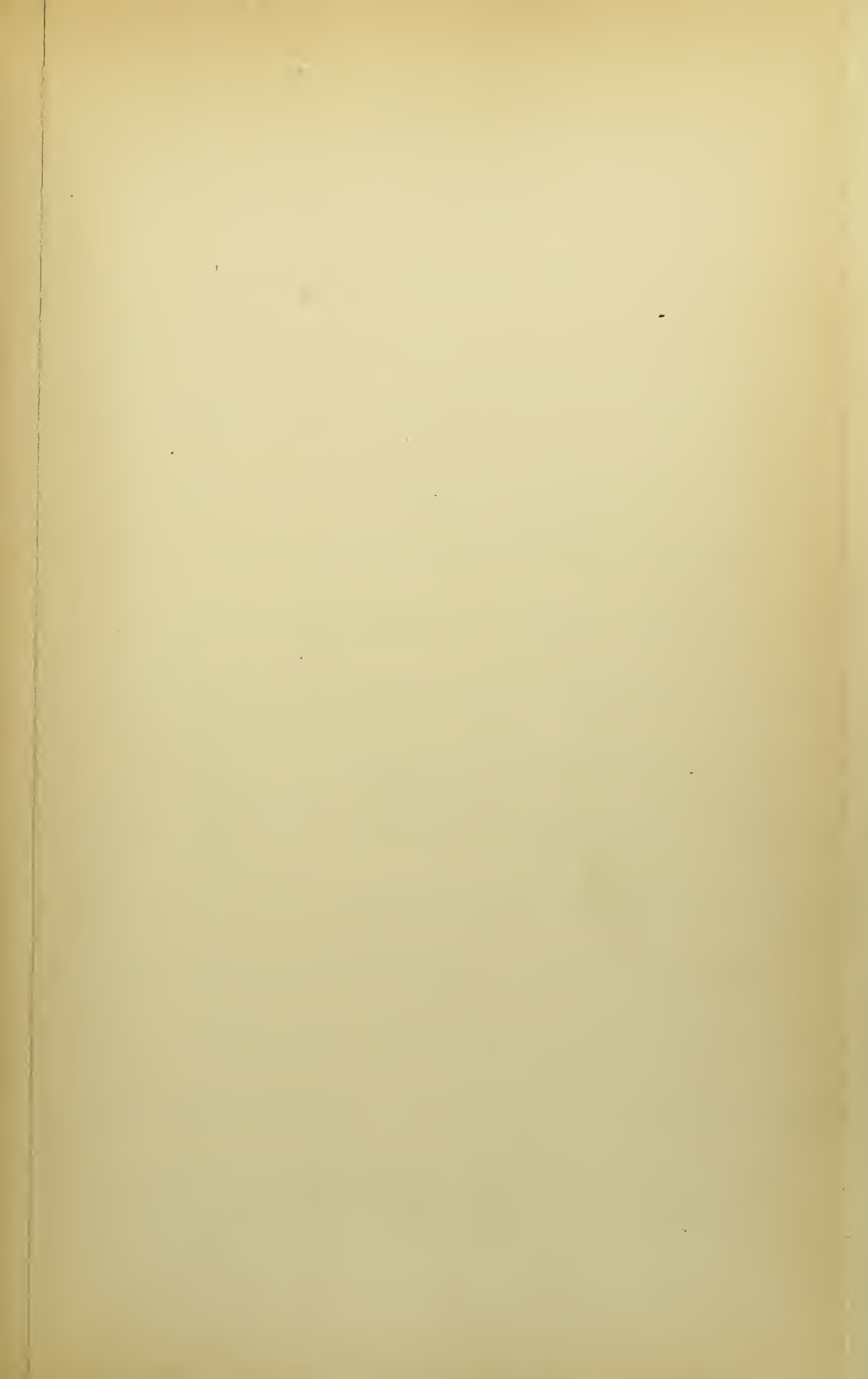
PHENO-SULPHO-PHTHALEIN TEST.

Record..... Date.....
Examination No.....
Name..... Address.....
Age.....
Childhood and previous diseases.....
Family History bearing on Heart and Kidney conditions.....
.....
Patient complains of.....
.....
Diagnosis.....
.....
Wassermann test was..... Blood pressure.....

URINE EXAMINATION	PHENO-SULPHO-PHTHALEIN TEST
24 hr. elim. was.....	1st, hr. elim.....%
Sp. grav.....	2nd, hr. elim.....%
Reaction.....	3rd, hr. elim.....%
Color.....	
Albumen.....%	
Sugar.....%	
Indican.....	
Urea.....	
Casts.....	

REMARKS

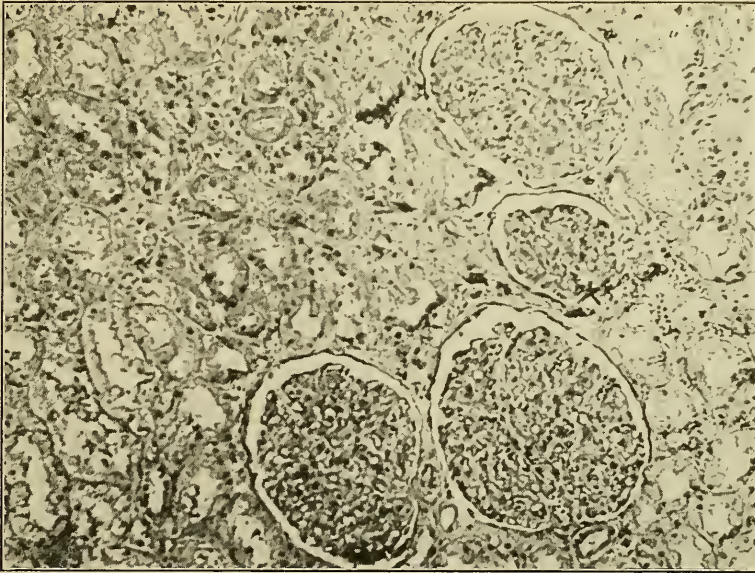
As subsequent tests were made, they were duly filed for a series of comparisons. From the above I have selected fifteen cases that are typical of the results obtained. These are duly tabulated on the Graphic Chart:



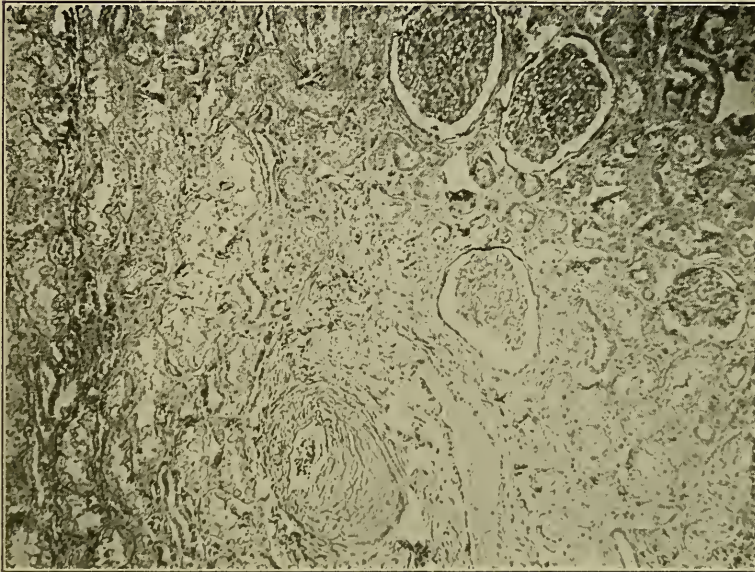
GRAPHIC CHART.
PHENO-SULPHO-PHTHALEIN TEST.

Name	Age	Occupation	Previous Diseases	Family History	Chief Complaint	Heart	Diagnosis	Wassermann	Blood Pressure	Urine Examination	Pheno-sulpho-phthalein Elimination	
Wyane.....	30		Scarlet fever, measles, whooping cough, rheumatism	Rheumatic	Œdema of legs, dyspnoea	Myocarditis, endocarditis	Cardio-renal	Not taken	120-170	1.018, straw, alk., no albumen or sugar	1st, hr. elim., 28% 2nd, hr. elim., 23% 3rd, hr. elim., 9% } = 60%	
Coady.....	62		Chr. alcohol. Typhus in early manhood	Alcoholic	Œdema of legs, dyspnoea, emphysema	Negative	Arterio-scl. and nephritis	Not taken	152	1.014, acid, tr. alb., many gran. casts	1st, hr. elim., 32% 2nd, hr. elim., 15.5% 3rd, hr. elim., 8.75% } = 56.25%	
Recinne.....			Small pox. Gen. at twenty, syphilis		Œdema of hands and feet, palpitation	Negative	Cardio-renal	Not taken		Not secured	1st, hr. elim., 45% 2nd, hr. elim., 25% 3rd, hr. elim., 13% } = 83%	
Winnie.....	35		Measles, chicken-pox, whooping cough	Negative	General weakness with Œdema of hands and feet, dyspnoea	Myocarditis	Cardio-renal	Not taken	115-150	Not secured	1st, hr. elim., 0 2nd, hr. elim., 40% 3rd, hr. elim., 10.75% } = 50.75%	
Walsh.....	41		Scarlet fever, measles and diphtheria	Alcoholic (three miscarriages)	Dyspnoea, double pyelitis		Double pyelitis	Not taken	150	1.006, acid, trace alb., few gran. casts	1st, hr. elim., 32% 2nd, hr. elim., 10.05% 3rd, hr. elim., 8% } = 50.05%	
Pender.....	55		Scarlet at twelve, rheumatism	Rheumatism, alcoholic	Spots before eyes, Œdema of hands and feet	Negative	Parench. neph.	Not taken	135	1.010, acid, alb. present, gran. casts	1st, hr. elim., 0 2nd, hr. elim., 7% 3rd, hr. elim., 4% } = 11%	
Lindesberg.....	46	Governess	Negative	Negative	Chronic headache, spots before eyes, night urin.	Hypertrophy	Parench. neph.	Negative	160	1.030, acid, alb. present, gran. and hyaline casts	1st, hr. elim., 60% 2nd, hr. elim., 12% 3rd, hr. elim., 5% } = 77%	
Harnis.....	56	Café business	Negative	Alcoholic	Dyspnoea, palpitation, cough, weakness	Double aort., incompetence	Cardio-renal	Not taken	185	1.024, acid, high alb., hyaline and gran. casts	1st, hr. elim., 10% 2nd, hr. elim., 9% 3rd, hr. elim., 14% } = 33%	Second Examination
Deperoo.....	20		Freq. tonsill. with heart trouble	Negative	Palpitation, Œdema of feet	Double mit. and aortic, incompetence	Myocardial	Negative	130-150	1.022, acid, large amount alb. with all varieties of casts	1st, hr. elim., 20% 2nd, hr. elim., 26% 3rd, hr. elim., 15% } = 61%	1st, hr. elim., 36% 2nd, hr. elim., 18% 3rd, hr. elim., 8% } = 62%
Cannocchi.....	45		Measles and whooping cough	Alcohol and heart trouble	Dyspnoea, Œdema of legs, weakness	Valvular dis., incompetence	Myocardial and valvular	Negative	100-120	Normal except high in indices	1st, hr. elim., 29% 2nd, hr. elim., 27% 3rd, hr. elim., 14% } = 70%	1st, hr. elim., 33% 2nd, hr. elim., 26% 3rd, hr. elim., 11% } = 70%
Sinnott.....	60		Diphtheria, measles, whooping cough, gon. syphilis	Negative	Palpitation, Œdema, aneurysm?	Specific endocard. and arteritis	Specific cardio-renal	Positive	170-200	1.030, acid, trace alb., hyaline and gran. casts	1st, hr. elim., 35% 2nd, hr. elim., 24% 3rd, hr. elim., 13% } = 72%	1st, hr. elim., 40% 2nd, hr. elim., 25% 3rd, hr. elim., 11% } = 76%
McKibby.....	48		Multiple abscess.	Alcoholic, Tubercular	Dyspnoea, Œdema of extremities	Myocarditis	Cardio-renal	Negative	190-200	1.010, acid, alb. present, high indican, hyaline and gran. casts	1st, hr. elim., 22.5% 2nd, hr. elim., 11% 3rd, hr. elim., 12% } = 45.50%	1st, hr. elim., 19% 2nd, hr. elim., 18% 3rd, hr. elim., 5% } = 42%
Burns..... (Normal case)	35	Laborer	Negative	Negative	Cellulitis of leg	Normal	Cellulitis of leg	Not taken		1.016, alk., no albumen, no sugar, no casts	1st, hr. elim., 27% 2nd, hr. elim., 60% 3rd, hr. elim., 5% } = 92%	Normal case.
*Schroeder.....	45	Laborer.....	Croup, rheumatism	Father alcoholic	Dyspnoea, Œdema, cyanosis, palpitation	Incompetence, mitral and aortic lesions	Chr. myocard. with borderline fibrillation	Negative	130-150	Unable to secure	1st, hr. elim., 20% 2nd, hr. elim., 30% 3rd, hr. elim., 16% } = 66%	1st, hr. elim., 27% 2nd, hr. elim., 27% 3rd, hr. elim., 12% } = 66%
*Johnson.....	42	Printer	Negative	Alcoholic	Dyspnoea, palpitation, Œdema of feet, hands and legs	Incompetent	Cardio-renal	Not taken	140	1.016, acid, trace alb., 1 per cent urea, high indican, gran. and byaline casts	1st, hr. elim., 11.5% 2nd, hr. elim., 13% 3rd, hr. elim., 4.5% } = 29%	

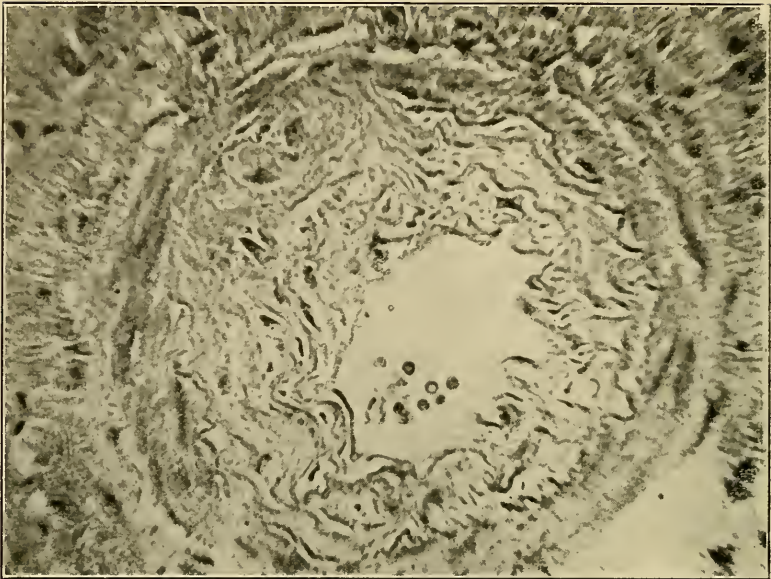
* See autopsy illustrations.



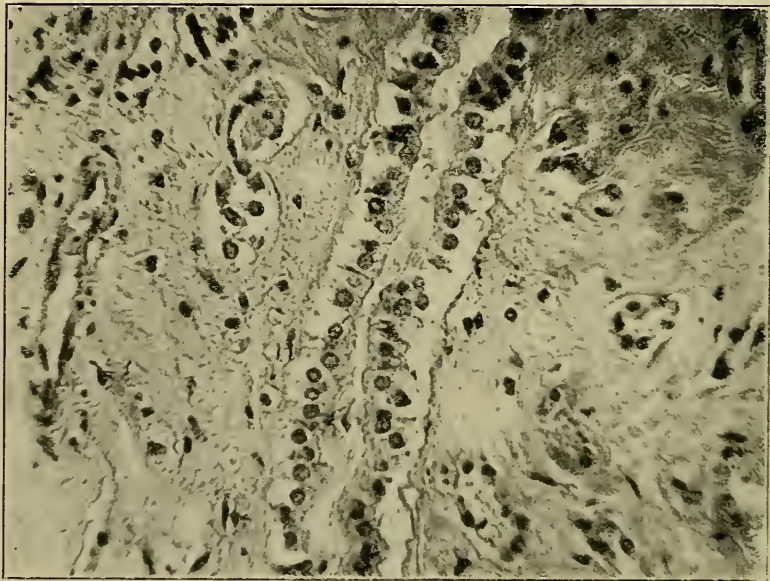
No. 1. (A) Schroeder. Chronic interstitial nephritis with tubules swollen and tufts encroached upon by the advancing fibrosis.



No. 1. (B) Schroeder. Chronic interstitial nephritis, with obliterating arteritis. Note size of lumen of artery and thickness of vessel wall.



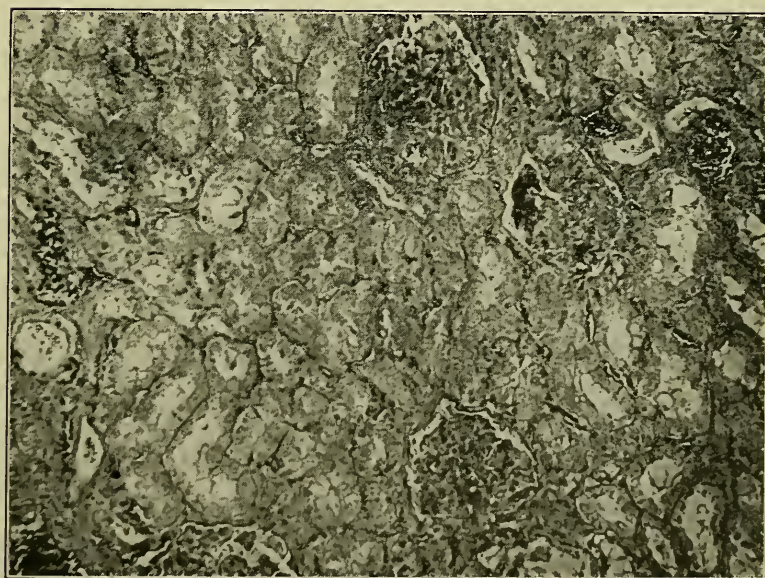
No. 1. (C) Schroeder. High magnification of blood vessel of kidney. Coats thickened. Interstitial nephritis.



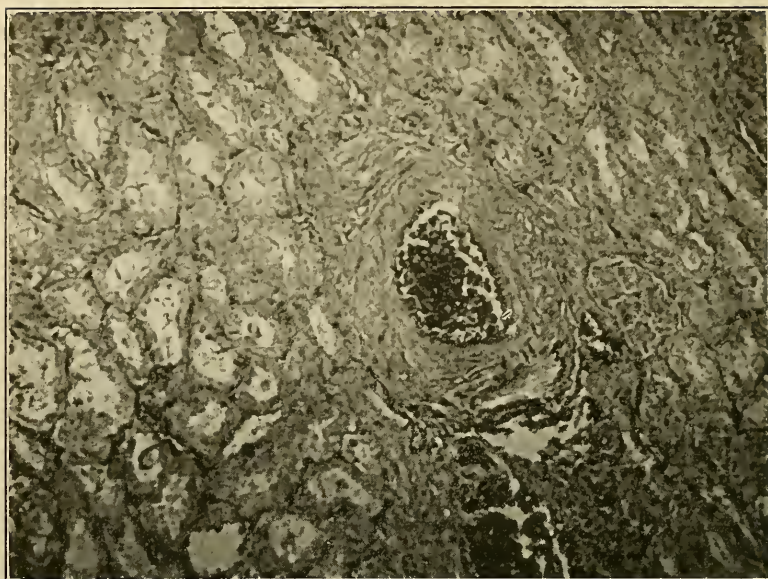
No. 1. (D) Schroeder. High magnification of kidney tubule showing irregularity with occlusion of lumen. Chronic interstitial nephritis.



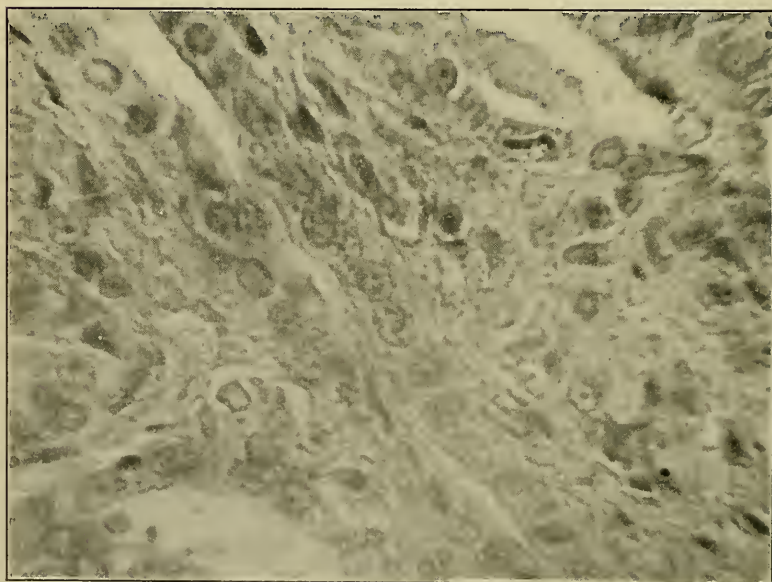
No. 1. (E) Schroeder. High magnification of kidney tuft fibrosis with contraction.



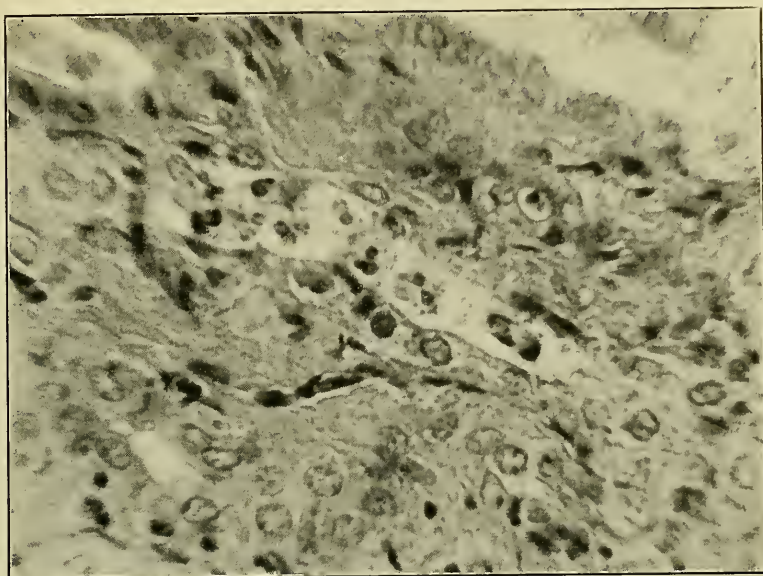
No. 2. (A) Johnson. Parenchymatous nephritis with colloidal degeneration of tufts and swollen tubules. Twenty-nine per cent color elimination.



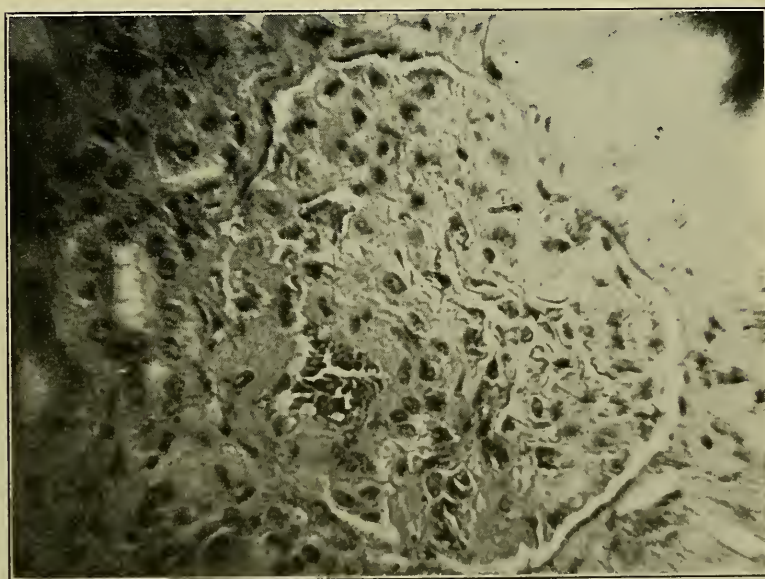
No. 2. (B) Johnson. Atheroma of artery of kidney in parenchymatous nephritis. Blood pressure, 140 mm.



No. 2. (C) Johnson. Showing tubule of kidney with partially obliterated lumen.



No. 2. (D) Johnson. Showing occluded tubule.



No. 2. (E) Johnson. Showing colloidal degeneration of the tuft. (High magnification.)

One of them used for control was the case of a normal man (Burns) with a cellulitis of the leg. His percentage showed 92 per cent. Most of them were true cardio-renal cases, having very serious cardiac and kidney lesions; compensation on the borderline, and bad valvular leaks; a class of case of great interest to the internist. The blood pressure observed varied widely and showed the close relationship existing between cardiac and renal function. The coefficient of heart, arterial system, and kidney seemed to be expressed in a mutual reliance upon this symptom.

In two instances the autopsy findings upon the kidney are interesting. Photo-micrograph No. 1 (Schroeder)—a true cardiac of borderline type, with rather frequent attacks of fibrillation—shows: No. 1 (a) Chronic interstitial nephritis with tubules swollen, and the tufts encroached upon by the advancing fibrosis. No. 1 (b) shows how far advanced is the arterio-sclerosis which has almost obliterated this vessel, and yet his blood pressure varied between 130-150 mm. His elimination was 66 per cent. upon two examinations. Cardiac dominating type.

Photo-micrograph No. 2 (Johnson), another cardio-renal, showed but 29 per cent. elimination, and presented marked evidences of No. 2 (a) parenchymatous nephritis and arterial changes. The tufts have partly undergone colloidal degeneration with obliteration, and in many instances the tubules are completely occluded; No. 2 (b) the arterial wall is very much thickened, particularly the media. Again with this advanced nephritis his blood pressure was 140 mm. Nephritic dominating.

In considering the possibilities of kidney function, we do not always give sufficient importance to Nature's rule of compensation. A series of studies on pathological kidneys convinces one of the constant effort on the part of the kidney to work as a unit, and that the particular pathological portion under the microscope does not always give a fair representation of other portions of the parenchyma which may be compensating by doing the added work thrust upon it.

Another result which seemed conspicuous was the part played by blood pressure. This symptom usually interpreted added work for the heart with reserve hypertrophy in whole or in part exhausted.

It is well to bear in mind that the true arterial changes found in advanced Bright's disease are general, and not confined to the kidneys. The kidneys unfortunately having a major part to play, have overshadowed the other viscera. In producing a more copious diuresis, only a temporary purpose can be served in warding off an approaching toxemia. The same fibrosis in the kidney is found generally distributed throughout the circulatory system, more so than anyone but the pathologist appreciates. For this reason the clinical picture is of paramount value in kidney elimination.

The test apparently was of value in paralleling the urine examination. In one case (Harms), a true cardiac-incompetent, after two weeks of digitalis, the output was increased from 33 to 73 per cent., but the general clinical picture showed this without any test.

We have purposely avoided any discussion of the merits as expressed by various authorities, owing to the fact that most of them have worked with the single object of approving or disproving the test per se. If it is accepted as a routine procedure and the clinical data carefully studied, at times it is suggestive of kidney disturbance. The urine, however, shows this with its more convincing pathological excreta. The various factors, however, entering into kidney elimination are so complicated we do not feel convinced that the mere passage of color through a kidney establishes any true data for pathological accuracy. There is a growing tendency on the part of the medical profession to lean too heavily upon tests of all kinds. The color test should come last—not first—and then as a check upon all the clinical findings. With the short time that this test has been available and the conflicting reports in the medical literature, it will be some time before its final accuracy is determined. In the meantime, it is not to be condemned until proven absolutely misleading or useless. Its greatest benefit may be in laying even greater stress upon more careful clinical study.

I wish to express my full appreciation to Drs. Reardon, Burdick, Wiley, and others of the House Staff whose painstaking analyses made these studies possible. Also to Drs. Potter, Brooks, and Shelby, of the Visiting Staff, who gladly turned over such clinical material as I might wish for the further study of this test.

COLON BACILLUS INFECTION IN MIDDLE-EAR DIS-
EASE, WITH THE USE OF AUTOGENOUS
VACCINES IN SUPPURATIVE OTITIS
MEDIA: REPORT OF CASES

BY

D. S. DOUGHERTY, M.D.,
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COLON BACILLUS INFECTION

The study of colon bacillus infection as a causative factor in disease has of late engrossed the attention of the profession and has occupied an important place on the programs of various societies. While papers have been read and cases reported by the internist, the proctologist, the gastro-enterologist, and the pediatrician, the otologist has not seemed interested, the infection having apparently not invaded his domain.

Taking into consideration the number of children in our tenement districts who suffer from running ears or eczema of the canal, neglected and dirty in habits, constantly rubbing or scratching their ears, one would be led to suppose that colon bacillus infection would be of frequent occurrence; but otological statistics make no reference to this microorganism as a factor in middle-ear infections or their complications. The history reports of two of our special institutions, as far as they could be arrived at; the history and laboratory files of the City Hospital; the laboratory statistics of Dr. J. H. Larkin, or those of Dr. F. M. Jeffries, and the research work of Haskin and Dwyer, furnish no instance of such infection.

It is therefore with particular interest that the Otological Division of the City Hospital finds itself able to make, as far as can be ascertained, the first report of a series of cases of aural infection by the bacillus coli communis. This lack of data may be due, of course, to the fact that patients of this class are usually treated in dispensaries or institutions in which the examination of the character of the infection—if any be made—is necessarily limited to the microscopic examination of a smear, the difference in diagnostic worth between a smear and a culture being often more distinct than a casual observer might imagine.

The colon bacillus not being a pathogenic organism per se, the criticism may be made that dust-carried or finger-conveyed bacilli may lie dormant on the surface of a normal canal and be present in aural infection as a concomitant but not contributing factor. This argument does not hold good, in view of the experience of Hall, Alberran, Ackerman, and others, which prove the virulence of the bacilli in various organs of the body, and

in the presence of other microorganisms. Dr. Larkin, also, has recently reported two cases in which colon-bacillus infection was the direct cause of death. An even more conclusive refutation of any such criticism lies in the fact that several of the cases cited showed the presence of the colon bacillus in pure culture.

There can be no question as to the active agent of the infection occasioned by the scratching of an abraded surface by a fecally contaminated finger.

A possible route for this character of infection is through the Eustachian canal during periods of severe vomiting, especially in the post-anesthetic state.

Case I. Female; age, 26; Bohemian; two months in the United States; married; one child, 15 months ago. Patient entered hospital by ambulance, with the following history:

Three weeks previous had experienced severe pain in the left external auditory canal and over the mastoid and temporal regions, but at no time referable to the middle ear. Two days later, she noticed a discharge from the canal, which continued for three days and then suddenly ceased. On this cessation of the discharge the pain became more intense, and the face and scalp became swollen.

Physical examination disclosed patient to be poorly nourished and anemic; left side of face and head edematous; left eye partially closed; a foul, purulent discharge from left ear; pyorrhœa, with badly decayed teeth; and tonsils enlarged and swollen.

Examination of the eyes was negative. Pupillary reaction good; no neuro-retinitis; no nystagmus.

Inspection of the ear showed the left canal to be reddened and swollen, and evidently the seat of an old furunculosis; tympanic membrane intact, slightly retracted, pinkish in color.

Temperature, 101.2 degrees; pulse, 104; leucocyte count, 19,000; polynuclears, 79 per cent.

On the same day, under general anesthesia, assisted by Dr. Harold Hays and House Surgeon Scudder, a large curvilinear incision was made, extending from the zygomatic region about one inch in front of the auricle, backward and downward behind the ear to the tip of the mastoid, evacuating an enormous amount of foul, greenish-yellow pus of a fœtid odor. The entire scalp was found to be raised from the bone, forming an abscess cavity which extended almost entirely over the temporal, parietal, and occipital regions. Directly under the necrosed mass of muscular tissue, which seemed to have been the first point of infective attack, there was complete dehiscence of bone, beginning at the juncture of the cortex and tegmen antri, and extending upward and forward, exposing an oblong section of the dura over the sphenotemporal lobe. This exposed dura was dark in color and covered with granulations.

A purulent discharge was welling up from the canal, and probing showed

necrosis of the bony posterior wall. The antrum was accordingly opened and the mastoid cells exenterated. The antrum was found filled with a grumous mass of pus and granulation tissue, and pus was found in the tip cells. The bony edges around the exposed portion of the dura were trimmed back until normal dura appeared, and the wound was dressed with iodoform gauze.

The question of opening the dura, for fear of cerebral abscess, was entertained, but was considered unwise on account of the virulent external infection.

A spinal puncture was made at this time, and the culture of the fluid obtained was later reported to be negative. Culture of the pus from the abscess and mastoid showed colon bacilli in pure culture.

The patient progressed comfortably from November 2d to 5th, the temperature ranging from 99 to 100 degrees.

November 6. Patient restless. Temperature rose to 102.5; pulse, 104; respiration, 24. Blood and spinal fluid taken for culture, with negative bacteremic results. Examination of the eye-grounds by one of the Visiting Ophthalmologists—Dr. Gilfillan—was also negative.

November 7. Temperature again rose from 100 to 102.5; pulse, 120; respiration, 26. Wound, when dressed, showed pus oozing from between the skull and dura. Dr. William Sharpe having expressed a wish to see the case, was invited to examine the patient, and under general anesthesia Dr. Sharpe and Dr. Hays went thoroughly over the case with me, both advising against opening the dura, no symptoms of brain abscess being present. Another culture of the pus showed colon bacillus in pure culture.

November 8-9. Patient restless at times; temperature, 99; pulse, 74-84.

November 11. Very restless, uttering sharp distressing cries. No Kernig; no Babinski; no nystagmus; eye-grounds normal. Seen by the Visiting Neurologist, who diagnosed the case as postoperative hysteria, with no organic lesion, and further operative procedure unwarranted.

November 12. Patient died at 8:40 P. M.

Autopsy showed abscess in the left temporal and occipital lobes. Bacteriological report: Colon bacillus present in pure culture.

Case II. Female; age, 9; United States. Was referred to my service with a history of having suffered from a severe earache on the right side, followed by a purulent discharge lasting three weeks, but which had suddenly ceased several days prior to the date of admission. Simultaneously with the cessation of the discharge, a swelling appeared behind the ear, which was gradually extending.

Inspection of the affected parts showed a large fluctuating mass over the mastoid and temporal regions; the auricle pushed forward, a normal tympanic membrane, and a collapsed posterior canal wall, with a small partially healed perforation in its middle third. A probe was passed through this, with slight pressure, and its withdrawal was followed by the exudation of a few drops of thick, foul-smelling pus. No systemic disturbance.

Temperature, 99; pulse, 86. Leucocyte count, 17,000; polynuclears, 70 per cent.

Under general anesthesia, the usual incision for the mastoid operation was made and carried well up over the ear, evacuating a large sub-periosteal abscess of half a pint of thick, malodorous pus. The posterior canal wall was found to be detached, and a grooved channel eaten into the cortex of the bone from inside the bony meatus to a saucer-shaped depression over the antrum. Here the bone had been corroded to a tissuelike thickness, and a second, wider groove channeled to the mastoid tip, which was entirely necrosed. Completion of the mastoid operation discovered the antrum free of pus or inflammation, but the lateral sinus partially exposed between the tip and the knee.

The post-operative course of the case ran to an uneventful recovery.

The bacteriological examination of the pus showed colon bacilli in pure culture.

Case III. Male; age, 20; single; United States. Entered the surgical division of the hospital for operation for ischio-rectal abscess, and on recovery was referred to my service for aural treatment. His ear affection had begun two months before entering the hospital, when he had noticed difficulty in hearing, followed by a purulent discharge from the right ear. Had had no pain, but now complained of throbbing in the ear and slight dizziness when walking. Had been treated in dispensaries without relief.

When seen by me (February 3rd), examination showed a right suppurative otitis media, with profuse foul discharge, entire loss of tympanic membrane, and presence of granulation tissue and polyps. Slight tip tenderness, and loss of both bone and air conduction. Pupillary reaction and accommodation normal; eye-grounds normal; slight nystagmus to right. Temperature, 100.2; pulse, 84; respiration, 22. Leucocyte count, 14,400; polynuclears, 82 per cent.

February 4, under ether anesthesia, assisted by Dr. Hays, a radical mastoid operation was performed. Upon removing the cortex, it was found that the middle ear, the antrum, and the middle cerebral fossa formed one continuous cavity, the bony partitions having been swept away. This cavity was filled with detritus and pus, thick in consistency and sickening in odor, and two small, hard, spherical masses of bone. The exposed dura was dark and covered with granulations. The posterior and tip cells were found filled with pus of the same character. The sinus was not exposed.

After thoroughly cleansing the wound, the dura was incised and a careful and thorough search was made for probable brain abscess, but with negative results. The wound was packed with iodoform gauze and dressed as usual. Culture of the pus showed large numbers of colon bacilli, a few staphylococci, and a gram positive bacillus.

February 6. Patient rested comfortably until February 6th, the second day after the operation, when he experienced a slight chill, and the temperature rose from 100 at 6 A. M. to 102.3 at 1 P. M. Next day, Febru-

ary 7th, he had a decided chill at 9 P. M., temperature rising to 104.4. Mentality good. No Kernig; no Babinski; no nystagmus. Wound dressed by Drs. Hays and Depping; looked clean. Blood and spinal fluid taken for culture.

February 8. Temperature fluctuated in sharp changes between subnormal and 104.2. Pulse, 106.

Report on bacteriological examination of blood and spinal fluid, negative. Leucocyte count, 16,400; polynuclears, 80 per cent.

February 9. A diagnosis of probable sinus or jugular thrombosis having been made on the sharp temperature curves, the patient was prepared for operation. Under ether anesthesia, assisted by Drs. Hays and Depping, the sinus was exposed from the torcular to the bulb, and was found normal in appearance and pulsating, but enormously enlarged, bulging out as soon as the bony covering was removed. The mastoid incision was then extended along the anterior border of the sternocleidomastoid to the clavicle, the carotid sheath was opened, and the external jugular vein exposed. This was found to be thrombosed just below the bulb for about one inch. After tying the collateral branches, the vein was excised in its entire length. The torcular end of the sinus was then compressed, the sinus opened, and the bulb curetted, it also being thrombosed. A free flow of blood being established from the torcular end, the sinus was closed by pressure of gauze pledgets, and the wound dressed, the neck wound being drained and strapped. Toward the end of the operation, 1,500 c.c. of normal saline solution and a half ounce of ergotol were infused, and repeated in four hours. Patient's condition poor; pulse, 144, small and irregular, and of low tension. Temperature, 101.2.

February 10-11. Temperature ranged from 99 to 100; pulse, 96 to 148, weak and irregular. Leucocyte count, 20,000; polynuclears, 90 per cent. Bacteremic examination of blood, negative; spinal fluid, normal; no cell count made, as specimen contained a few drops of blood. Culture of pus taken from the wound at this operation showed colon bacillus and streptococci, moderately hemolytic. Thrombus contained disorganized hyaline material, fibrin, red blood cells, and blood pigment; no organisms.

February 12. Patient died at 1:40 P. M. No autopsy permitted.

Case IV. Female; age, 7. Entered hospital with diagnosis of gonorrheal vaginitis. Transferred to the otological service December 12, with double purulent otitis media, culture showing Friedlander bacillus. Treated by irrigation and autogenous vaccine. January 30, right ear dry. Some discharge from left ear. March 24, adenoids and tonsils removed. Discharge from left ear increased. Middle ear filled with granulations, and posterior wall of canal ulcerated. After considerable delay, consent was obtained and a radical mastoid was performed. Culture showed colon bacillus and bacillus pyocyaneus. Recovery was uneventful.

Case V. Female; age, 14. Entered otological service March 27, with history of suppurative otitis media since January. Tonsils and adenoids

removed three years ago. Radical mastoid performed by Dr. Hays. Culture of pus showed colon bacillus and streptococcus viridens. Patient discharged under care of Dr. Hays.

Case VI. Male; age, 3. Entered surgical service for operation for tubercular cervical adenitis. Transferred to otological division suffering from purulent otitis media, left ear. Bacteriological report, bacillus coli communis predominating; a small amount of streptococcus mucosus and a gram positive bacillus.

Case VII. Male; age, 3. Entered service January 20th, suffering from double otitis media suppurativa. Treated with irrigations, consent for operation not obtained.

March 15. Both ears discharging profusely; tympanic membrane absent; cellulitis of left external auditory canal. On March 17, consent having been obtained, a double radical operation was performed. Biological character of the pus: Bacillus coli communis in pure culture. Patient discharged cured.

Case VIII. Male; age, 9 months. Entered service from dermatological division March 7, with large perforation of left drum and profuse discharge of pus. Culture showed colon bacillus and streptococcus hemolyticus. Before treatment could be rightly instituted, the patient was transferred to the Riverside Hospital, having apparently developed diphtheritic laryngitis.

Case IX. Male; age, 4 years. Transferred from ophthalmological service April 25th, with right otitis media purulenta, and treated with irrigations and vaccines. Culture showed presence of colon bacillus and a non-identified gram positive bacillus.

Case X. Male; age, 42. Case of acute mastoiditis complicating chronic suppurative otitis media. Culture of pus from both middle ear and antrum showed colon bacillus and bacillus xerosis.

Case XI. Male; 11 years. Entered hospital January 15th, suffering with cerebral meningitis. Transferred to the otological division April 26th, with double suppurative otitis media. Bacteriological report: Colon bacillus and staphylococcus aureus. Treated with vaccines, with excellent result.

Case XII. Male; 4 years. Entered hospital with diagnosis of acute mastoiditis. Profuse discharge, and edematous mass behind left ear. Examination and operation revealed furunculosis of canal and perforation of the posterior wall, with consequent establishment of a large subperiosteal abscess filled with foul pus. The cortex was eroded but not perforated. Continuation of the operation showed antrum and tip free of pus. Bacteriological report: Colon bacillus in pure culture.

It is of interest to note that Cases I, II, and XII are analogous in that the colon bacillus was found in pure culture and that the initial point of entrance of the infection was through the canal wall, the three cases differing only in the severity of the destructive process. In Case VII, in which the bacillus was in pure culture, the point of inception in the left ear was also furuncular.

In Case III we have primarily a chronic otitis media with slight staphylococcic infection dormant until lighted up by the virulence of the colon bacillus which was conveyed direct from the rectal abscess. The streptococci found with the colon bacilli at the time of the second operation were probably due to secondary infection.

Case IV, in which the Friedlander bacillus had disappeared and the ear cleared up under local treatment, must have been due to a subsequent direct colon infection acquired while in the hospital. The remaining cases were mixed infections, in two of which the colon bacillus was largely predominant.

AUTOGENOUS VACCINES

When in 1910 Dr. Nagle first reported her experience in the use of autogenous vaccine in the treatment of suppurative affections of the middle ear, it occurred to me that the City Hospital offered a wonderful field for this character of experimental work, and the laboratory authorities were asked to collaborate in a series of cases. Unable to obtain their assistance, a small quantity of stock vaccine was procured and several cases were treated, but with no appreciable results. Later, the changes in the pathological department and the improvements in the laboratory facilities enabled me to secure the necessary coöperation, and during the past eighteen months the pathologist has rendered every assistance and entered enthusiastically into the work.

Owing to the loss of certain of my personal notes and the laxity with which the clinical records have been kept at times, I am not able to report all the cases treated—some twelve or fifteen being missing. Believing that the results from any small number of cases would hardly be considered as in any way authoritative, the following twenty-four cases are presented simply as a preliminary report.

These cases were in no sense selected cases, but were taken by the house staff at random from the various hospital wards, the only selective element being the likelihood of their remaining in the hospital long enough to insure sufficient observation.

Where there was evidence of necrotic bone tissue, operative procedure was recommended, and the vaccines were used only as secondary or post-operative treatment. In fact, it is now my rule to treat all post-operative mastoids with autogenous vaccines, experience showing that the period of healing is thus considerably shortened.

Although no absolute conclusions as to the efficacy of this treatment can be drawn from these experiments, the results so far obtained warrant its continuance. Two important lessons have been taught by the comparative failure of our early cases, one in the technique of obtaining the pus for culture; the other in the preparation of the vaccine.

At first the pus was obtained by means of a cotton swab passed through the canal, with a resultant contamination of the vaccine with extraneous

bacteria from the canal—sterilization of the canal with antiseptic irrigations having been found likely to too thoroughly cleanse the middle ear. The method now employed is to wipe the canal dry and then sterilize it by placing in it for several minutes a pledget of cotton saturated with alcohol. The pus is then obtained by the use of a platinum loop. In case of a small perforation, suction may be made with a Siegel otoscope.

To secure the best clinical results, it was found necessary that the vaccine should not only contain the correct strains, but that the exact relative proportion of each bacterial element should be maintained.

No untoward symptoms occasioned by the vaccines were observed, even when used in large increasing doses, and the reaction, if any, was unnoticeable.

Case I. Age, 6. Purulent otitis media of right ear, of several months' duration. Friedlander bacilli. Began vaccine treatment December 7, giving one million every third day, 12 injections in all. Ear dry on January 20, when patient was discharged cured. Seen six months later; ear dry.

Case II. Age, 5. Chronic suppurative otitis media, left ear; two years' duration. Streptococcus and staphylococcus aureus. Treated with autogenous vaccine in increasing doses—one to ten millions—for several weeks, without success. Radical mastoid performed, but suppuration recurred and persisted for two months. Staphylococcus aureus. Vaccine treatment, one million every third day for ten doses. Patient discharged, ear having been dry for two weeks.

Case III. Age, 3. Suppurative otitis media, right ear. Colon bacillus and streptococcus mucosus. Treated for twelve days, with appreciable results in the quantity and character of the discharge. Then transferred to the surgical division. (Case not followed, as patient died from ether pneumonia.)

Case IV. Age, 3. Double otitis media. Colon bacillus in pure culture. In this case a double radical mastoid operation was performed in conjunction with the vaccine treatment. One million every three days, between October 17 and November 1. Discharged cured, November 12, the ear having been dry for twelve days. No suppuration to date (May 30).

Case V. Age, 8. Chronic suppurative otitis media, right ear. Streptococcus viridans, of one month's duration. Began vaccines October 28, starting with one million and gradually increasing to ten. November 14, ear dry; taken from hospital by parents. No report since.

Case VI. Age, 7. Suppurative otitis media, left; and peritonsillar abscess, left. Streptococcus viridans in pure culture. Vaccines, one million, increasing to ten, March 5 to 15th, when ear ceased to discharge, and patient left hospital. Mother now reports ear well.

Case VII. Age, 9. Otitis media suppurativa, left. Streptococcus hemolyticus. Treated from February 8 to March 27—one to ten millions. Improved. Still slight discharge at times.

Case VIII. Age, 34. Left suppurative otitis media of one year's dura-

tion. Wassermann reaction, positive. *Staphylococcus aureus* and *micrococcus catarrhalis*. Vaccines every day for five days—one, two, four, six, and ten millions. Discharge stopped. Patient left hospital. No further history obtained.

Case IX. Age, 22. Right suppurative otitis media. *Streptococcus hemolyticus*. Treated March 21 to 30, in increasing doses to 100,000,000. Left hospital with scarcely perceptible discharge.

Case X. Age, 9. Left otitis media suppurativa. *Staphylococcus aureus*. March 23 to April 8. Tonsils and adenoids removed before commencing treatment. Left hospital improved. Discharge has since ceased.

Case XI. Age, 14. Colon bacillus and *streptococcus viridans*. Treated without results. Radical mastoid done, and vaccine treatment continued. Still has some discharge.

Case XII. Age, 4. Right purulent otitis media, of several months' duration. Colon bacillus and gram positive bacillus, unidentified. Left hospital improved.

Case XIII. Age, 40. Suppurative otitis media, right, four weeks' duration. Paracentesis had been performed prior to admission. *Streptococcus* and *staphylococcus*. Twenty millions every other day for five injections. Cured.

Case XIV. Age, about 38. Unhealed mastoid, from another institution. *Staphylococcus*; twenty million every other day; eight injections. Discharged cured. Has remained so to date.

Case XV. Age, 35. Suppurative otitis media; six weeks' duration, following paracentesis for acute condition. *Bacillus mucosus capsulatus*. Sixteen injections of twenty millions each. Discharged, ear dry. Remains so to date.

Case XVI. Age, 26. Chronic suppuration of left ear, of three months' duration. *Staphylococcus aureus*, very hemolytic. Placed on vaccine treatment April 3, one hundred millions; April 5, two hundred millions; April 10, five hundred millions; April 16, seven hundred millions; April 20, eight hundred millions. Left hospital April 24, with ear fairly clean; very slight discharge. Returned May 4, suffering from acute mastoiditis. Operated and again placed on vaccine, in smaller doses. Still in hospital.

Case XVII. Age, 20 months. Double suppurative otitis media. *Staphylococcus aureus*. Vaccines as follows: April 17, one million; April 23, two millions; April 30, two millions; May 11, twenty-five millions; May 19, twenty-five millions. On May 30, right ear dry; left, very scanty discharge.

Case XVIII. Age, 2 years. Double suppurative otitis media. *Streptococcus viridans* in pure culture. Autogenous vaccines as follows: March 12, fifty millions; March 15, sixty millions; March 17, eighty millions; March 19, eighty millions. Ears dry and continue so to date.

Case XIX. Age, 3 years. Unilateral otitis media suppurativa. *Streptococcus hemolyticus*. Had been treated locally for several months without

result. On March 10th and 25th, and on April 2nd, received fifty millions. May 30, ears dry.

Case XX. Age, 2 years. Double otitis media. *Streptococcus hemolyticus*. Vaccines as follows: February 21, five millions; 28th, ten millions; March 5th, ten millions; 7th, ten millions; 9th, twenty millions; 12th, fifty millions; 25th, one hundred millions; April 4th, four hundred millions. Ears dry. Continue so to date—May 30th.

Case XXI. Age, 7. Double suppurative otitis media. *Staphylococcus aureus*. May 4, one hundred millions; May 7, one hundred millions; May 11, three hundred and thirty millions; May 13, three hundred and thirty millions. No discharge, May 30.

Case XXII. Age, 25. Left otitis media purulenta. Paracentesis of drum done, and mastoid operation recommended but refused. Four weeks later, commenced administration of autogenous vaccine. Twenty millions every third day for one week, and forty millions every other day for two weeks. Discharged cured. No trouble since.

Case XXIII. Adult. Right otitis media purulenta. *Streptococcus hemolyticus*. On December 31 had symptoms of acute mastoiditis but refused operation. Autogenous vaccines administered every other day, with very appreciable results. Discharge lessened and became thinner, and mastoid symptoms disappeared. Evidently, however, only held in abeyance, as patient left hospital January 21st and was subsequently operated upon in another institution.

Case XXIV. Adult. Left otitis media suppurativa. Treated with vaccines, twenty millions every other day for two weeks, beginning December 27. Discharged January 17. Ears dry. No report since.

REPORT FROM THE PATHOLOGICAL DIVISION

BY

DR. J. H. LARKIN

DIRECTOR

REPORT FROM THE PATHOLOGICAL DIVISION

BY DR. J. H. LARKIN,
Director

Perhaps no one hospital offers such advantages as the City Hospital with the great amount of anatomical material at hand. During the past year there has been from the 500-odd deaths 225 autopsies performed. The greater number of these cases have been of the rarer kind, which demands special investigation. All such material as is thought advisable is kept for future reference, and special examinations are made of the tissues of such cases. The work of this department has been handicapped by the lack of assistance, there being a greater amount of work now than the present force of assistants can handle. It is hoped, however, that during the coming year the number of qualified workers will be increased. The varied character of the cases coming to autopsy covers a wide range. This is especially true, not only of the visceral anatomy, but also of the pathological anatomy of the brain and spinal cord. During the past year several publications have been made on such cases, and at present others are approaching completion. It is hoped that during the year 1915 other pathological reports will be published. The distribution of such pathological material is as follows:

All such material showing the newer and rarer forms of disease is made use of in the Pathological Department for the instruction of students on the house staff, and especially with the clinician who has had the individual under care during life. Already there is a well-stocked and select museum of such cases, and upward of some 600 individual mounts are to be found in the Pathological Museum.

The library of the Pathological Department, however, is not up to a high standard, being maintained out of the private purse of the Director. There is necessarily a shortage of such periodicals and books as are constantly to be found in similar laboratories connected with medical colleges. It is hoped that the Department of Charities will maintain hereafter a well-equipped library, containing all modern periodicals and books relating directly to pathological anatomy, pathological chemistry, and bacteriology.

The filing and reporting of all data from the several departments has been maintained by a special worker. The work consists of the recording of all autopsy findings, the filing and reporting of all serology reactions and bacteriological results promptly to the several wards of the Hospital.

Attached to this report will be found a general statement of the work accomplished during the past two years in the departments of Serology and Bacteriology.

The Pathological Staff consists of the following:

One Director, Dr. John H. Larkin, who has supervision of all pathological, serological, chemical, and biological work.

Three assistant pathologists, as follows:

1. Assistant Pathologist, Dr. I. J. Levy, who has entire control of all serological and bacteriological work.

2. Assistant Pathologist, Dr. Leon H. Cornwall, who has entire charge of all histological, pathological, and such special work as may be designated to him by the Director.

3. Assistant Pathologist, Dr. Wm. W. Hala, who was recently assigned to this laboratory from one of the other departmental laboratories. Dr. Hala is in charge of special pathological and bacteriological work; also directing the laboratory in clinical pathology.

4. Pathological Chemist, Mr. Louis Pine, who has entire charge of the Department of Pathological Chemistry, and all special research work in this connection.

5. Two Pathological Interns, who are assigned to the Pathological Laboratory from the Alternate List of Interns of the City Hospital, and who are under the direction of the Director.

6. Stenographer and typist, having special care of all records in the laboratory; filing and maintaining a card catalogue of all results in the serological, pathological and bacteriological departments, and the distribution of same to the several wards of the Hospital.

7. Pathological Technician, who has charge of all technical work, such as cutting of sections; keeping same labelled and classified for ready reference, and preparing all stains and reagents used in the laboratory.

8. Serological Technician, who is under the direction of the Serologist.

9. Bacteriological Technician, who prepares all media used in the laboratory, and who is under the direction of the Director.

10. Two morgue men.

YEARLY REPORT OF WORK DONE AT THE STRECKER MEMORIAL LABORATORY, CITY HOSPITAL, FROM JANUARY, 1912 TO JANUARY, 1913.

Number of deaths.....	501
Number of autopsies performed.....	220
Coroner's cases.....	58
Gross pathological specimens preserved and mounted for museum purposes.....	183

BACTERIOLOGICAL EXAMINATIONS: .

—Blood cultures	
—Widal reactions	
—Throat cultures	
—Animal injections	
—Sputa	
—Pus, etc.....	1084

Wassermann reactions.....	5,497
Gonococcus fixations.....	153
Autogenous vaccines prepared.....	51
Surgical specimens sectioned, stained and diagnosed.....	175
Typhoid vaccines prepared for stock.....	1
Leukocytic extracts prepared.....	34
Cerebrospinal fluid examined and cultured.....	34

Special chemical analyses as follows:

—Nitrogen.....	215
—Urea.....	86
—Ammonia.....	65
—Uric acid.....	182
—Sodium chlorid.....	165
—Albumin.....	1
—Fat.....	2
—Dextrose.....	114
—Gastric contents.....	3
—Sarcosylactic acid.....	2
—Sulphur.....	4
—Phosphorus.....	1
—Calcium.....	4
—Oxalic acid.....	2

TOXICOLOGICAL ANALYSES:

—Arsenic.....	1
—Mercury.....	1
—Lead.....	3

Summary

Total number of Bacteriological, Serological and Pathological processes during the year.....	7,192
Total number of Chemical and Toxicological analyses.....	849
Grand Total.....	8,041

In giving a report of the serological and bacteriological work done in the laboratory a detailed account of the amount and character of the work is only incidental. Of greater importance is a consideration of methods and facilities necessary to obtain the best results. Our purpose has been two-fold. Firstly, to assist the clinician in diagnosing the cases in the hospital; secondly, to attempt to collect the data of the various examinations for future study.

That we have succeeded in the first instance is shown by the great demand made on the laboratory by the clinicians of the hospital. The work up to the present time has consisted only of that requested by the attendings. Before May, 1911, less than 200 bacteriological examinations were done; the serology, apparently, had been neglected. From May, 1911, to Jan., 1913, and from Jan., 1913, to Jan., 1914, there has been surprising demands made, as the following tables will show:

	May, 1911 to Jan., 1913	Jan., 1913 to Jan., 1914
Wassermann reactions.....	4,800	5,500
G. C. fixations.....	123	153
Bacteriological examinations.....	602	1,084
Vaccines.....	23	131

Our attempt at collecting data, unfortunately, has not met with the same success. In the laboratory, however, a recent system has finally been installed which is of great assistance, not only in keeping accurate notes of the findings in our examination, but is valuable in aiding us make a systematic study of any portion of the work. Unfortunately, only in the hands of a competent man is such a system practical. Several times names and reports have been so confused as to render our work worthless. At present, however, there is no possible criticism in this department.

The interpretation of the value of laboratory work entirely depends upon the study of the cases upon which the various clinical examinations have been made. Our work is only partly done if in a hospital with 900 beds we confine ourselves to routine work necessary in diagnosing the various maladies. With such abundant material found in the wards the opportunity for study should receive the serious attention of those actively engaged in the laboratory. This fact has been thoroughly recognized. To accomplish it means organization. We have attempted it partly in the serological investigation of syphilis, though we were seriously handicapped in obtaining accurate and definite data on the various patients in the hospital. An exhaustive study of these cases meant the assistance of men directly in charge of the patients; namely, the interns. With a view of obtaining accurate clinical data printed slips outlining a short history and the important facts of each case were to be filled out by the interns, and were to accompany each specimen of blood or spinal fluid sent to the laboratory for diagnosis. It took weeks to institute this clerical work. Our efforts have met with only partial success. Either the slips accompanying the specimen of blood or spinal fluid intended for a Wassermann diagnosis were not filled in with the data asked for, or the data given was inaccurate. The reason is quite evident. Though the men on the intern staff are absolutely competent to meet every requirement demanded of them, the staff is not properly organized to work as a unit. Demands made by the laboratory on the various services require instructions to be given to several men, or several groups of men. A Chief of Staff, as a resident physician who was responsible for the work of the interns would be not only a distinct advantage to the hospital service, but of great assistance in aiding the laboratory in its work.

For the past two years separate data sheets accompanying each blood or spinal fluid for Wassermann reaction were used. Recently, however, it occurred to us that a record which incorporated a short résumé of the history of the case, the serological diagnoses arranged consecutively and the treatment outlined, would be both practicable and valuable to the clinician, and of great service to the serologist. With the assistance of our stenographer such a chart was devised, and is now in print. The following is a copy of this Wassermann Reaction Chart:

WASSERMANN REACTION CHART

Reference No.....

Patient.....

Address.....

Age.....

Nationality.....

Ward	{	Male.....
		Female.....

Date of first admission to Hospital.....

Date of second admission to Hospital.....

Date of third admission to Hospital.....

Date of first discharge.....

Date of second discharge.....

Date of third discharge.....

NOTICE: This chart must accompany each specimen of Blood or Spinal Fluid submitted to the Laboratory for diagnosis. The result of each examination will be recorded on the chart, and the chart then returned to the proper ward.

NOTICE. This chart is to be forwarded to the Laboratory after discharge of the patient.

RECORD OF SPECIMENS SENT TO LABORATORY

No.	Specimen	Tube Number	Diagnosis	Date Sent	Date Reported	Approval of Pathologist
1	Blood					
	Sp. Fl.					
2	Blood					
	Sp. Fl.					
3	Blood					
	Sp. Fl.					
4	Blood					
	Sp. Fl.					
5	Blood					
	Sp. Fl.					
6	Blood					
	Sp. Fl.					
7	Blood					
	Sp. Fl.					
8	Blood					
	Sp. Fl.					
9	Blood					
	Sp. Fl.					
10	Blood					
	Sp. Fl.					

HISTORY SHEET

Hereditary History.....
Date of Primary..... Incubation Period.....
Character of Primary.....
.....
Date of Secondaries.....
Character of Secondaries.....
.....
Date of Tertiaries.....
Character of Tertiaries.....
.....

CLINICAL DIAGNOSIS

Syphilitic Origin.....
Non-syphilitic origin.....

IMPORTANT SYMPTOMS

.....
.....

.....M. D.

House Phys. or Surgeon

SALVARSAN
TREATMENT SHEET

No.	Dosage	Kind	Method	Date
1				
2				
3				
4				
5				
6				
7				
8				
9				
10				

TREATMENT OTHER THAN SALVARSAN OR MERCURY

.....
.....
Doctor's Observation.....
Final Observation on Discharge.....

MERCURY
TREATMENT SHEET

No.	Dosage	Kind	Method	Date
1				
2				
3				
4				
5				
6				
7				
8				
9				
10				
11				
12				
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The first page of this chart contains space for the serial reference number, which is supplied by the record room of the laboratory, and which is placed on a card with the name of the patient, and filed alphabetically in a card-index for ready reference. The first page of the Wassermann Chart also provides space for data as a patient's first admission, and also for re-admission to the hospital.

The second page of the Wassermann Chart provides space for a consecutive record of the serological diagnoses, to be referred to quickly.

The third page is designed for a short résumé of specific infection with present clinical diagnosis as to syphilitic or non-syphilitic genesis and other important symptoms.

The fourth sheet is purely a sheet to show the details of each reaction.

The last two sheets, the Salvarsan and Mercury Treatment Sheets, are self-explanatory—they are designed to show the number of treatments of salvarsan and mercury, and the method of administration in each case.

SEROLOGY

About 15,000 Wassermann reactions have been completed to date. In the year 1913, 5,000 examinations were reported on 2,000 cases, an average of over two reactions per patient. The work was begun in June, 1911. The system employed was the one originally suggested by Wassermann, and closely followed with the exception of substituting various antigens which were considered an improvement on the syphilitic liver. The antigens prepared from luetic livers were found only fairly satisfactory since—

(1) The proportion of positive cases fell far short of the number examined.

(2) And the various antigens were not uniformly good. The antigens prepared from the alcoholic extract of guinea-pig heart, as recommended by Landsteiner, were then used. Two important facts were here noted:

(a) A greater proportion of undoubted syphilitic cases gave positive results.

(b) It was soon apparent that more vigorous treatment was indicated to render a positive reaction negative.

Noguchi's acetone partition antigen had much to recommend it, so it was decided to incorporate a third antigen along with the other two. Several thousand cases were done in this manner. The general deductions can be tabulated as follows:

(1) In secondary syphilis the proportion of positive reactions with the three antigens was fairly uniform, about 95 per cent. of such cases giving a positive reaction.

(2) In tabes and paresis, on the other hand, a greater percentage of positive results was obtained with the guinea-pig heart antigen.

(3) Treatment resulted in a negative reaction with the specific liver and acetone partition antigens very early, depending, of course, on the individual case. It was evident then that no advantage was to be gained by continuing with these two antigens, the guinea-pig heart antigen giving far the most satisfactory results. It was, however, far from ideal for these reasons:

(1) After treatment, and with a negative reaction, the discharged patient in many cases returned with clinical manifestations of syphilis, his serum again giving a positive reaction.

(2) A high percentage of patients suffering with tabes gave negative reactions.

Several men in Germany had introduced the reinforcing of normal

and syphilitic tissues with cholesterin. Swift, of the Rockefeller Institute, advocated its use, and he reported favorably on such antigens. With the hope of further improving the diagnostic value of the test several kinds of antigens have been reinforced with cholesterin. The past year each specimen sent to the laboratory for a Wassermann reaction has been reported according to the result based upon three antigens. The antigens used are:

(1) Human heart, normal or syphilitic, extracted with absolute alcohol at 37 deg. C. for 10 days, to which 0.4 per cent. of Kahlbaum's cholesterin has been added.

(2) Guinea-pig heart, extracted with absolute alcohol for five days at 37 deg. C., 4 hours at 60 deg. C., also with 0.4 per cent. cholesterin.

(3) Same as (2), but without the addition of cholesterin.

The system corresponds closely to that suggested originally by Wassermann. The total volume is 5 c. c. and the sheep's hemolytic system is employed. Complement and hemolysin (sheep amboceptor) are titrated each time. Complement is discarded if 1 c.c. of 1:10 dilution of the serum does not contain at least 2 units. The hemolysin is so diluted that 1 c. c. of the dilution contains over two, and not more than three, units.

The antigens must comply with the following:

(1) The anti-complementary point must be at least four times the antigenic titration.

(2) It must contain at least five antigenic units when titrated with a serum taken from an acute secondary case of syphilis. It usually is 10, and often higher.

(3) It should not vary when compared with the previously used antigens. At least 50 different sera are used. This, at least, entails uniform results.

The serological results with the above procedure have been thoroughly and exhaustively studied. We have learned that it is a marked improvement over our former methods; but that the serological diagnosis is not always an absolute and dependable diagnosis. In other words, there is a small percentage of positive cases which give a negative fixation, depending, of course, upon the type of syphilis; and undoubtedly negative sera occasionally give positive results. Hence, the importance of interpreting every serological reaction. This phase of the work has been sadly neglected by the serologist. At a later date, a technical report will be given on the value of the reaction in diagnosing active and latent lues, considering its interpretation in every phase of the disease; its value as a guide to treatment and the serious objection of accepting a serological diagnosis as final, especially when done by an inexperienced worker.

The examination of the spinal fluid in syphilis has gained almost equal importance with the serological examination of the blood. The early work was confined to cases with distinct nervous lesions. Since, in competent hands, the withdrawal of the fluid is attended with practically no danger to

the patient, and with very little discomfort, in many cases not any, the study of the spinal fluid from patients suffering with various manifestations of syphilis is now within the scope of every laboratory, and has given the clinician valuable information. That the syphilographer and medical diagnostician consider the spinal fluid findings absolutely essential in basing an opinion on the presence of an organic lesion even more so than the neurologist is partly shown by noting the number of such examinations done at the laboratory, and a consideration of the character of the various cases. In the year dating from January 1, 1913, to January 1, 1914, 184 spinal fluid examinations were reported, which is 9 per cent. of the total number of Wassermanns done that year, by far the greater number of positive reactions being from the neurological service (70 per cent.). From January, 1914, to June, 1914, however, 166 spinal fluids were examined, representing over 20 per cent. of the total number; about 70 per cent. of the positive findings in this six months came from the medical and syphilitic services.

The report of the spinal fluid findings include the serological examination, the cell-count and the presence of globulin estimated by the Noguchi and ammonium sulphate methods. Heretofore, the cell-count was left to the various members of the intern staff. This proved unsatisfactory. It was soon evident that several months of technical training was essential for any individual to make an accurate count. At present such work is left to one of the laboratory staff with much better results. Immediately after withdrawal of the fluid the cell-count is done and the globulin estimated. The fluid is diluted with a methyl violet acetic acid solution and counted in a Fuch-Rosenthal chamber. Staining the cells in this way is a distinct advantage over the unstained method and insures greater accuracy. A study of the morphology of the various types of cells found is an important factor in the examination. It is attended with some difficulty, the routine methods offering a number of technical difficulties. At present it is proposed to adopt a method similar to the following:

Centrifuging the fluid and embedding the residue in paraffin; cutting serial sections and staining. It is also proposed to include a description of the structure of the various cells in the report sent to the wards.

The interpretation of the laboratory findings of spinal fluid examination in the light of the clinical manifestation of the disease is now being thoroughly investigated. To facilitate this work it was necessary to arbitrarily designate the results of the examinations as, (1) "POSITIVE SPINAL FLUID," (2) "DOUBTFUL SPINAL FLUID," (3) "NEGATIVE SPINAL FLUID."

- (1) POSITIVE SPINAL FLUIDS: $\left\{ \begin{array}{l} \text{(a) Positive Wassermann} \\ \text{(b) Cell-count over 12} \\ \text{(c) Increased globulin.} \end{array} \right.$

- (2) DOUBTFUL SPINAL FLUIDS: Absence of any one of the above factors.
(3) NEGATIVE SPINAL FLUIDS: Absence of the three factors.

Health, and other sources, a polyvalent antigen was prepared according to the method recommended by McNeil and Schwartz. Our results with the latter antigen were more definite and satisfactory. Since only a few cases of acute gonorrhea are admitted to the Hospital and the material at hand consists mostly of gonorrheal joints, cardiac conditions, and other chronic G. C. affections, the value of the reaction could only be estimated in these types of the disease. The one great source of difficulty is obtaining an accurate and definite clinical diagnosis, especially in chronic joint involvement. The fixation method of obtaining a diagnosis of gonorrheal infection deserves serious attention. The well-known and popular method of diagnosing gonorrhea, both before and after treatment, by stained spreads, should be thoroughly condemned for its inaccuracy and the serious consequences possible following an opinion based on this procedure.

Some attention has been given to streptococcus fixation tests in streptococcal infection. About 50 strains of streptococcus have been collected from various sources, and antigens prepared. The work will occupy at least another 10 months before completion.

BACTERIOLOGY

The demand for bacteriological examinations during the past year has so taxed the department that it was necessary to call upon volunteer workers to assist in the routine work. With the consent of the attending staff, two men are appointed each year from the incoming intern staffs for service in the Pathological Department for a period of one year. The system has proven a success. The work is popular with the men, and after a few months' training they can be called upon to assist in the various bacteriological examinations.

The preparation of the media is a very important factor in obtaining good results. For a detailed and accurate bacteriological diagnosis it is essential not only to have media that are reliable, but to be well stocked with the various kinds necessary for differential diagnosis. It is surprising in a well-equipped laboratory what little attention is given to media, and still more remarkable what definite bacteriological conclusions are recorded without studying thoroughly the cultural characteristics of a microorganism. Several technicians well recommended proved to be absolutely inefficient in preparing media that were worth using. With this experience one was trained by the workers in the laboratory, with the result that the medium is accurately titrated, well stocked, and is uniformly good.

The medium is titrated with phenolphthalein to 0.9 acid or neutral. Two kinds of meat are used for infusion: lean beef and young veal. The meat is extracted overnight in the ice-box chest and next day with heat. The agar is cleared for one hour in the autoclave at 15 lbs. pressure, and finished in the Arnold. The sterilization is done in the Arnold for one-half hour for three successive days. The following is a list of the media always

on hand. "Plus" indicates acidity; "N" indicates neutral, and "Minus" alkalinity.

2% BEEF AGAR SLANTS, 0.9 PLUS	$\left\{ \begin{array}{l} \text{Plain} \\ \text{Glucose} \\ \text{Ascitic plain} \\ \text{Ascitic glucose} \\ \text{Blood plain} \\ \text{Blood glucose} \end{array} \right.$	2% BEEF AGAR STABS, 0.9 PLUS	$\left\{ \begin{array}{l} \text{Plain} \\ \text{Glucose} \end{array} \right.$
2% VEAL AGAR SLANTS, N.:	$\left\{ \begin{array}{l} \text{Plain} \\ \text{Glucose} \end{array} \right.$	2% VEAL AGAR STABS, N.:	$\left\{ \begin{array}{l} \text{Plain} \\ \text{Glucose} \end{array} \right.$
BEEF BOUILLON:	$\left\{ \begin{array}{l} \text{In 100 c.c. Flasks:} \\ \text{In Tubes:} \end{array} \right. \left\{ \begin{array}{l} \text{Plain, plus 0.9} \\ \text{Glucose, plus 0.9} \\ \text{Calcium carbonate glucose, N.} \\ \text{Plain, plus 0.9} \\ \text{Glucose, plus 0.9} \\ \text{Calcium carbonate glucose, N.} \end{array} \right.$		
LITMUS SERUM:	$\left\{ \begin{array}{l} \text{Dextrose} \\ \text{Inulin} \end{array} \right.$	BEEF BOUILLON 0.9 PLUS IN FERMENTATION TUBES:	$\left\{ \begin{array}{l} \text{Glucose} \\ \text{Saccharose 2\%} \\ \text{Lactose} \\ \text{Maminute} \end{array} \right.$

Conradi Drigalski

2% AGAR FLATS:	$\left\{ \begin{array}{l} \text{Beef agar plain, 0.9 plus} \\ \text{Veal agar plain, N.} \\ \text{Veal agar plain, N., salt free} \end{array} \right.$
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The clinicians on the medical service make frequent requests for blood cultures, not only in suspicious cases, but many times to eliminate any possibility of a bacteremia. Two hundred and sixty blood cultures have been taken the past year. Of these two hundred and ten were negative on unidentified cases; thirty-three on lobar pneumonia, 18 per cent. of which were positive; five typhoid cases, 65 per cent. of which were positive. Streptococcus was isolated in nine cultures; three were cases of puerperal sepsis; one, pneumonia. In five the origin of the infection was unknown. Colon was isolated in two cases; one patient suffering with an ulcer of the rectum, in the other the original focus was unknown.

The blood is cultured at the bedside. Needles and syringes are sterilized in the laboratory. Two agar stabs, one plain, the other glucose, for plating; and three bouillon flasks, one plain, one glucose, and one calcium carbonate glucose, are taken for each culture. Ascitic fluid is often added to one of the agar stabs. A small amount of blood is also drawn into a sterile tube containing a few crystals of calcium oxalate and this is cultured in the laboratory. A report is given at the end of twenty-four, forty-eight and seventy-two hours.

In the year 1913 thirty-one autogenous vaccines were prepared for various infections and stock cultures and stock vaccines kept for future use. Two methods of standardization are used in the laboratory: the Wright method and counting directly in a chamber, the emulsion diluted

with a stain in a red counting pipette. A report of the therapeutic value of the various vaccines is left to the clinicians on the various services.

All material for bacteriological examination is sent to the laboratory for diagnosis, with the exception of spreads stained for tubercle bacilli, or the Gram stain for intracellular gonococcus, or meningococcus. This is done by the interns in the Clinical Laboratory.

The agglutinating, iso-agglutinating, and hemolytic serum tests are left to the bacteriological workers. The Widal is done with two strains of typhoid: on an old culture, the other recently isolated; two strains of paratyphoid, an alpha and beta, with the macro- and microscopical method. The macroscopical method is by far the more satisfactory, though the less popular one.

In closing it is difficult for me to express fully my thanks to the officials who have so greatly aided me in the reorganization of the Pathological Laboratory. I have been the recipient of many kindnesses from the Commissioners of Charities, who, though in public life, have lent me their aid and support in a most magnanimous manner. The Medical Board has supported me in every possible way, as shown by their presence and interest in the laboratory work. To my personal assistants, Dr. I. J. Levy, Dr. Leon H. Cornwall and Dr. William W. Hala, Mr. Louis Pine and Mr. Howard Kay, I am under great obligation for aid and for many helpful suggestions, which they so kindly gave.

THE PREVENTION OF FETAL ASPHYXIA BY THE
ADMINISTRATION OF OXYGEN TO
THE MOTHER

BY

GEORGE PEASLEE SHEARS, M.D.,
ATTENDING OBSTETRICIAN

THE PREVENTION OF FETAL ASPHYXIA BY THE ADMINISTRATION OF OXYGEN TO THE MOTHER

BY GEORGE PEASLEE SHEARS, M.D.,
Attending Obstetrician

During my service at the City Hospital in 1912 I treated a number of toxemic cases by the free use of oxygen. This was done upon the hypothesis that the toxemia of pregnancy, or, as it is more properly called, the pre-eclamptic condition, is a result of suboxidation. Both the theory and the treatment have enlisted the sympathetic attention and interest of colleagues at the City Hospital and elsewhere.

Many incidental confirmations of this theory have occurred to me or have been suggested by others, and the result of considerable recent experience both at the City Hospital and elsewhere, which I hope to publish later, together with the stimulating interest of various medical friends, and of valued correspondents whom I have never had the privilege of meeting, have encouraged me to persevere in this work. I now believe not only that oxygen is a valuable adjunct to the treatment of both toxemia and eclampsia, but that its occasional use in pregnancy is desirable and beneficial, serving as a prophylactic and invigorator even in cases approximately normal, and especially in those cases in which the patient is obliged to remain in-doors a large part of the time. I also believe that it will do much to relieve the slight subjective dyspnea, the weakness, oppression, and discomfort, so common in cases probably mildly toxic, but not far enough advanced to present typical symptoms. It is almost always subjectively agreeable to the patient.

Oxygen has been empirically and occasionally used in eclampsia as a heart stimulant and for the relief of the cyanosis and respiratory obstruction attending the convulsions. It has also been injected into the breasts upon the hypothesis that eclampsia is due to some toxin elaborated in the process of milk secretion.

As far as I have been able to ascertain, however, its systematic and continued use as an agent to combat suboxidation in toxemia and eclampsia has not been hitherto advised or practiced.

In the milder cases of toxemia I allow the patient to inhale oxygen for five or ten minutes every two or three hours, and in the more severe cases every hour or even half hour. In eclampsia it is given almost ad libitum.

Of course I do not claim that all cases of toxemia can be cured by oxygen alone, or that oxygen is the only agent that does good in these cases.

I advocate its use as the most direct method of obtaining relief. It interferes in no way with the employment of those measures already mentioned, which experience has shown to be of unquestionable benefit, and which I always employ.

Nor do I believe that we should stop with these. Basing my practice upon the theory already outlined, I advise the use of every possible dietetic, hygienic, and medicinal resource for the promotion of the processes of oxidation. To enumerate them all here would carry us too far. Of especial importance as prophylactics are life in the open air, the avoidance of foods whose conversion into energy taxes the oxidative functions, frequent bathing, and moderate, judiciously regulated exercise, from the beginning of pregnancy. Iron, proverbially beneficial in this condition, is an oxygen carrier, and the same thing may be said of calcium, the use of which has recently been advocated. Dr. Bullowa, of the New York Polyclinic, has suggested that the beneficial effect sometimes noted as the result of the administration of thyroid extract is due to the stimulating influence of the latter upon oxidation. Every physician soon becomes familiar with the bad effect upon pregnant women of ill-ventilated rooms and crowded places.

And so one might go on almost indefinitely. On the whole it would perhaps be better to speak of the treatment here suggested as the oxidation treatment, rather than the oxygen treatment.

Dr. Homer Wakefield, of this city, who has written extensively and profoundly upon the problems of metabolism and physiological chemistry, and to whom I am indebted for many valuable suggestions, confirms my belief that all the dietetic and hygienic measures that have been found beneficial in the toxemia of pregnancy are promoters of oxidation.

The object of this communication, however, is not to discuss the treatment of the toxemia of pregnancy, but to call attention to certain immediate effects of oxygen upon the fetus which were observed in the course of my studies upon this subject, and which, I think, have hitherto escaped attention.

In the first place, the inhalation of oxygen by the mother appears to increase the rapidity of the fetal heart. I have before me the notes of eleven tests made at the City Hospital. Each test consisted in the inhalation of oxygen for five minutes by a pregnant woman at or near term, the fetal heart being counted before and after inhalation. Fetal movements were also observed and uterine contractions noted. In order to eliminate the personal element I did not do the counting myself, but entrusted the task to one of my assistants. The rapidity of the fetal heart sounds was increased in every case. In two cases the increase was so small as to be practically negligible, two and four beats respectively; in a third it was eight beats, and in the remainder it was from sixteen to twenty-five beats. The average increase was about fifteen beats. The real increase was somewhat greater than is here indicated, since the inhalation of oxygen appears to excite uterine contractions, and in at least four cases the counting was

done during a contraction. It is a matter of every day experience that the fetal heart beats more slowly during a contraction.

Another new and curious fact noted during these experiments is that oxygen increases the frequency and activity of the fetal movements. Out of nine tests it was found that the fetal movements were increased in five. In three of these they were marked simply "increased" and in the remaining two "markedly increased." In the case of one patient near term and since delivered, the fetus underwent remarkable contortions, but without harm.

Why do the fetal heart sounds become more rapid, and the fetal movements more active, under the influence of oxygen? Certainly not because of a general stimulation of the maternal organism. Drugs given by the mouth or hypodermically do not ordinarily affect the fetal heart. The fetal and maternal circulations, however, are really one, and if oxygen enters into chemical combination with the maternal blood, how can the blood of the fetus escape its influence?

Now if we can administer oxygen to the fetus, and if the fetus responds symptomatically to that administration, is it not plain that we can do much in the prevention and treatment of ante-natal asphyxia? The normal slowing of the fetal heart during contractions usually does no harm, but if the labor is much prolonged or if the contractions become tetanic in character, the fetus will sooner or later show the effect of the diminished supply of oxygen. During a uterine contraction there is, of course, a corresponding diminution of the placental area. The fetus thus receives less blood and consequently less oxygen. In our experiments at the City Hospital the fetal heart sounds were increased in frequency during the contractions. Therefore it would seem a wise precaution to administer oxygen to the mother in cases of prolonged second stage, in partial separation of the placenta, or whenever slowness or irregularity of the fetal heart sounds indicates impending asphyxia.

I am aware that there are many sources of error in counting the fetal heart, but the fact that in eleven cases an increase was noted in every case, that the average increase was about fifteen beats per minute, and that in some cases it was as high as twenty-five beats per minute, and that in a large number of cases the fetal movements were increased, sometimes markedly so, seems to indicate that the inhalation of oxygen by the mother is not without its effect upon the fetus.

In two subsequent tests conducted in the same manner the heart rate was increased ten beats per minute and fetal movements were observed.

Shortly after this, one of my house surgeons whose name I do not now recall had an opportunity of witnessing the stimulating effect of oxygen upon the fetus during a difficult version. With his hand in the uterus he distinctly felt fetal movements immediately following the administration of oxygen to the mother.

Since the above experiments were made I have continued the use of

oxygen in difficult labor and in obstetric operations with a view to preventing pre-natal asphyxia, and with apparently favorable results. Several of my colleagues have had the same experience.

On the basis of these experiments I have been in the habit of advising that whatever anesthetic is used in obstetric operations it be given with a large admixture of oxygen in order to minimize the danger of fetal asphyxia from the anesthetic as well as from causes incident to the operation itself.

At the present time there is in one of the wards of the City Maternity a patient suffering from placenta previa. She was an ambulance case, had been bleeding for some time before admission, and was beginning to show symptoms of acute anemia. The cervix barely admitted three fingers. A foot was brought down and traction upon this brought the half breech into the cervix which, however, was hard and not very dilatable. The bleeding now ceased but forcible extraction or other operative procedure was not deemed advisable. The fetal heart rate was 136. Oxygen was administered, whereupon the rate rose at once to 160. About forty-five minutes elapsed between the bringing down of the breech and the delivery of the head. During this time moderate traction was made upon the fetal leg in order to control the bleeding. Meanwhile oxygen was administered and the fetal heart was constantly watched. Shortly before delivery it became very rapid (190) but remained of good quality. The child was in excellent condition and no measures of resuscitation were required.

Another patient also in the hospital at this writing was admitted suffering from profound toxemia. The usual treatment, rest in bed, milk diet, catharsis, etc., was supplemented by frequent inhalations of oxygen and marked improvement followed. The treatment, however, including the inhalations, was continued. At the end of a week I questioned her as to the immediate effects of the inhalations. In reply, she stated among other things that during or after these she "felt the baby kicking." I had said nothing about the child nor was I thinking of the effect of oxygen upon the fetus at the time.

This unsolicited and unsuggested testimony of the patient, a very intelligent woman, confirming as it does the observations mentioned above, is to my mind highly significant.

I desire to emphasize the fact that in order to eliminate the personal element I did not do the counting of the fetal heart myself. It was done in every instance by a member of the house staff, but not always by the same member. The fetal movements too were observed by different members of the house staff, with the single exception of the case referred to above, in which they were made many times by the patient herself.

Another patient now under treatment in a private hospital gave similar testimony, and adds the interesting information that when the oxygen is given by one of her nurses, who is evidently a convert to this method of treatment, the effect upon the fetus is much more marked than when it is

given by the other nurse, who is less prodigal in its use and is evidently working in the interest of hospital economy.

I have no further hesitation in making the claim that it has been definitely proven that we can administer oxygen to the fetus and that the fetus responds to its administration.

AN UNUSUAL CASE OF FRIEDREICH'S ATAXIA

BY

WILLIAM STEINACH, M.D.,
ATTENDING NEUROLOGIST

AN UNUSUAL CASE OF FRIEDREICH'S ATAXIA

BY WILLIAM STEINACH, M.D.,
Attending Neurologist

While hereditary ataxia or Friedreich's disease is scarcely of sufficient rarity to merit reporting, the following case presents several features which differentiate it from those hitherto described.

The patient, J. D., 15 years of age, white; born in New York City, was admitted to the Neurological Pavilion of the City Hospital, May 20, 1913. His mother died of pulmonary tuberculosis 14 years ago; one brother died of a complaint denominated eczema by the patient, at the Skin and Cancer Hospital, having been there one week prior to his death. He has one brother alive and well.

His father is alive and for the last eight years has suffered from tabes dorsalis. His history briefly is as follows: He had a chancre 25 years ago which was not followed by any secondaries. Eight years ago he began to walk with uncertainty, had numbness and tingling in his legs; shooting pains and bladder symptoms. When examined recently he showed all the ordinary symptoms of tabes. He had Argyll-Robertson pupils, absent knee-reflexes, Romberg, delayed sensation and typically ataxic gait.

Aside from the father no member of patient's family has suffered from any nervous or mental disease. Careful questioning of the father failed to elicit any evidence that there had been any epilepsy, alcoholism, or any neurological disorder in the immediate or collateral branches of the family.

When three years of age the patient had an attack of eczema involving the head and arm. He escaped the ordinary diseases of childhood and there is nothing of note in the history until he reached the age of 5, when he narrowly escaped being run over by an express train. For about a year after this incident patient was extremely apprehensive and nervous and afraid of being alone or in the dark.

At the age of ten the patient noticed difficulty and unsteadiness in walking. This unsteadiness spread upward from the legs, and shortly after extended gradually to the trunk and arm muscles. At this time also there was noticed some difficulty in talking, but mentally there was no deterioration.

For the past year and a half the patient has been practically helpless on account of his extreme ataxia and must be dressed and undressed like a child. He cannot get about, and must be taken out in a wheeled chair.

There have been no bladder or rectal symptoms.

Mentally the patient is normal, although he appears somewhat defective on casual notice.

PHYSICAL EXAMINATION

General Appearance—White male, poorly nourished and poorly developed. Musculature is generally atrophied, but there is no atrophy of any definite muscle groups. As the patient sits in his wheel-chair his body and head are markedly inclined to the right side. There is a right lateral scoliosis of the spine with corresponding deformity of the chest.

Eyes—The pupils of both eyes respond to light and accommodation and the consensual reflex is present. There is no paralysis of any of the ocular muscles. There is marked nystagmus.

Ears—Normal; no Darwinian tubercle.

Mouth and teeth show no abnormality.

Tongue protrudes in the median line; there are slight choreiform twitchings of the tongue when protruded.

The abdominal reflexes are present on both sides.

Extremities—The arms are markedly ataxic; muscular power diminished on both sides.

The legs are considerably atrophied; movements very ataxic. When assisted to walk he spreads his feet apart and walks in typically ataxic fashion. His knee reflexes are absent on both sides and he has a very marked Babinski of both feet. There is no ankle clonus on either side. There are no sensory changes. Heat, cold, pain and touch are normal. His speech is ataxic—typically the "hot-potato" speech.

His blood was examined for the Wassermann reaction on May 27, 1913, and was found to be weakly positive. On August 6, 1913, it was negative.

Spinal puncture done on August 4, 1913, revealed a clear fluid; there was increased pressure; a normal amount of globulin; no Fehling reduction and a cell count of 2.

The spinal fluid showed a doubtful Wassermann reaction.

The urinalysis made August 5, 1913, showed a specific gravity of 1.022 acid reaction; no albumen; no sugar; no casts.

This case is a typical Friedreich's ataxia and is of interest for several reasons.

In most of the cases of Friedreich's disease which have been reported the same disease has occurred in several members of the family or its collateral branches. In two other cases recently in our wards a brother and sister were affected in each instance.

The peculiar interest of the case, however, is in the possible etiological relation to lues and in the fact that the father was suffering from undoubted *tabes dorsalis*.

TUBERCULOUS MASTITIS IN AN ACTIVE BREAST

BY

NATHAN W. GREEN, A.M., M.D.,
ATTENDING SURGEON

TUBERCULOUS MASTITIS IN AN ACTIVE BREAST

BY NATHAN W. GREEN, A.M., M.D.,
Attending Surgeon

The rarity of this condition is my excuse for publishing a report of this case. The patient, M. O'C., age 22, housewife, married, born in Ireland, was admitted to the maternity service of the City Hospital February 24, 1914, and was delivered of a normal child March 3, 1914.

While she was thus in the maternity ward, on the service of Dr. Franklin A. Dorman, I was requested to see her by the house officer, Dr. Croft. I found the patient well nourished, recently delivered and presenting upon her left side a number of scars, which are well shown in the accompanying photograph, Fig. 1. The family history is as follows: Her father and mother are living and well; four brothers and sisters are well and living in Ireland; three infant brothers have died. Previous to coming to this country she had been well. In 1912 she came to the United States and shortly after her arrival she had four teeth extracted, two upper and two lower molars on the left side. The teeth had been in bad condition for one year previous. Two months after the teeth were pulled an abscess followed, as she said, behind her ear, and then all the lymphatic involvement which is shown by the scars in the photograph followed. First, the lymphatics of the neck were affected, then the left axilla, then over the sternum and then the involvement of the breast took place. She had noticed this last point two or three months before the baby was born. An abscess broke after childbirth. The patient was transferred from Dr. Dorman's service to mine on March 16, 1914, and the breast was removed the following day by Dr. Depping, my House Officer, by turning up a flap. The convalescence was slow but uneventful, her temperature remaining at 98.4 and pulse from 74 to 90. After the breast was removed another lymph node became involved and then broke down under the right axilla. This was subsequently opened and is now nearly healed. There were no further complications, except that the wound of the breast amputation is still partly open, but is healing steadily. The diagnosis which was made before operation was confirmed by the pathological report of Dr. John H. Larkin, Director of the Strecker Memorial Laboratory. It is as follows: "Microscopical examination showed tuberculous mastitis in an active mamma; stained section showed tubercle bacilli."

At present, no pulmonary involvement is detected on examination. Her



Fig. 1. This picture shows the extensive scarring due to the breaking down of the tuberculous lymph glands; and by the nipple is shown the seat of a true tuberculous mastitis.



Fig. 2. Micro-photograph of a section of the pathological specimen.

general condition is good and her surgical condition appears well on the road to recovery.

The interesting features of this case are the clinical picture, the extensive scarring with repair, showing a splendid example of overcoming the infection, and the history of the infection arising after the removal of carious teeth.

I am indebted to Dr. Arthur Sherman, Assistant Attending Surgeon, for the excellent photograph of the patient, and to Dr. John H. Larkin, Pathologist, for the micro-photograph of the section of the pathological specimen, Fig. 2.

DENTAL SEPSIS: ITS RELATION TO THE SYSTEM

BY

C. N. B. CAMAC, M.D.,
ATTENDING PHYSICIAN

DENTAL SEPSIS: ITS RELATION TO THE SYSTEM¹

BY C. N. B. CAMAC, M.D.,
Attending Physician

The cases upon which the following observations are based presented purulent infection in and about the teeth. These cases were studied in private practice and in the medical service of the New York City Hospital. The terms used in literature to indicate this condition are oral sepsis, Riggs' disease, pyorrhea alveolaris, dental sepsis, dental abscess, necrosis, etc. By the internist these terms are very loosely used, and one must turn to works upon dentistry to get any accurate information of the nature and extent of the *local* process. Even in such works, however, there is no adequate explanation of the relation of these variously situated foci to each other. Works on internal medicine contain as yet no comprehensive discussion of this subject, and its causative relation to some of the most destructive systemic processes (arthritis, endocarditis, etc.), a relationship which we know to be of primary importance in the consideration of the etiology, diagnosis, and therapeutics of these conditions. It is the duty of the internist to familiarize himself with this focus of pyogenic infection *in its early stage* and to coöperate with the dentist as with the surgeon in the eradication of foci of pus in any part of the body. The teeth, especially at the pulp, may, like the tonsillar, peritonsillar, and deep urethral tissues, harbor pyogenic organisms and even small amounts of virulent pus without local manifestations, although systemic manifestations having apparently no relation to this source appear from time to time, and may reach a crippling or fatal degree. It is this latent or subtle quality in the local infection which causes it to be frequently overlooked or discounted by the internist. Such foci can often be detected by radiographic examination only. In one instance (Case I) the only manifestation was a slight crepitation of the left temporomaxillary joint, which was unaccompanied by pain or tenderness. There was slight ear pain, for which the patient consulted an aurist. This, and the unusual sound in opening and closing the jaw, was the only complaint. The radiograph showed a pulp abscess. The tooth was removed and a pure culture of *Streptococcus viridans* was obtained from the pulp, socket, and gum, and the blood gave positive complement fixation for the same organism. There was no fever

¹ Presented at the meeting of the Association of American Physicians held at Washington, D. C., May, 1913 (see Transactions of the Association of American Physicians, 1913), and extracted from the American Journal of the Medical Sciences, February, 1914, No. 2, vol. cxlvii, p. 186: to which is now added a case observed in the New York City Hospital.

at any time and no leukocytosis. Vaccines were prepared from this tooth, but were not used at the time, as conditions did not seem to justify doing so. A few months later general arthritis developed and the autogenous vaccine was immediately employed, with marked benefit, but not with entire disappearance of the arthritic manifestations. The blood still showed positive fixation for the same organism. On further search, another tooth was found, with small pus focus, and on removal of this there was rapid improvement, with ultimate disappearance of the arthritis. The manifestations of arthritis were pain on motion, active and passive, and slight tenderness on deep palpation, but no redness or local fever. The knees, ankles, wrists, elbows, and small joints of the hand were involved. The temporomaxillary joint was, however, the only one permanently damaged, and in consequence the crepitation persists (see case reports, Case I). This case well illustrates the slow, steady absorption, with destruction, unless the focus is detected and the process checked. It is always possible in the protracted cases in which no removal of the focus or other checking of the infection is carried out, for new foci to develop in the joints or soft tissues. Under these circumstances the condition may become progressive, consequent to but independent of the original focus and often hopelessly destructive to the joints or visceral tissues. The persistence of the temporomaxillary joint signs in the case just cited makes it possible that here may be a new focus of infection which may appear in other localities later. Several such new foci, even though the original focus has been drastically treated by removal of the tooth and by curettement, make the clinical problem one of difficulty, disheartening the patient and baffling the physician. Vaccines under these circumstances are the only rational therapeutic remedy. This case demonstrates the importance of close coöperation between dentist and internist. This obligation to coöperate rests as much with the dentist, if not more so, as with the internist. The dentist may find a tooth abscess with which, in most instances, he deals mechanically. Later the internist sees the same case, and though he may associate the history of the diseased tooth with the systemic disturbance which he now finds, it is often impossible, at that late date, to accurately correlate the two processes. The dentist in most instances does not make cultural studies of the pus, and thus the only opportunity of detecting the invading organism and of preparing vaccines may be lost. If, as seems likely, vaccines prove to be the only certain means of combating these destructive pyogenic infections in their advanced stages, the loss of the opportunity to procure this important therapeutic agent would be a serious and culpable omission.

Setting aside the question as to whether the dental involvement is the cause or effect of a general sepsis, the clinical fact remains that the material procurable from such foci frequently offers the only, and often brief, opportunity to identify the invading organism and of procuring materials from which vaccines may be made. I prefer to emphasize this feature of the subject, which is of so great diagnostic and therapeutic importance, rather

than that of cause and effect, which, as a question of etiology, involves problems possibly unsolvable. The cases upon which this report is based have been studied in coöperation with Drs. M. I. Schamberg, Alexander Currie, W. D. Tracy, Harold S. Vaughan, dentists of New York City, three of whom are also graduates in medicine. Among the radiographs accompanying this article I have included those of cases which have come under the personal observation of these dentists, through whose courtesy I am enabled to reproduce them here.

In using the term coöperation I do not mean to infer that the patients were told to consult the dentist merely, but I accompanied them to the dentist's office and studied with him the radiographs and local conditions. I believe such consultation to be as necessary as those common between surgeon and internist. When a pyogenic dental infection requiring liberation of the pus existed, arrangements were made for bacteriological specimens to be procured at the dentist's office, and later, if conditions indicated, a specimen of blood for culture and complement fixation tests was taken. A certain number of dentists are in accord with this practice; indeed, a small number have gone beyond the medical practitioner, and without his coöperation are studying cases with just such thoroughness as outlined above. There is, however, among the dentists a large class of dental tinkers, who practice upon the easy persuasibility of the public. A large mass of mechanical work is thus done upon the teeth with often an entire neglect of the destructive focus of pus. A specific instance of this occurred in Case II. In this case there were (see case reports, Case II) areas of pus from which *Streptococcus viridans* in pure culture was obtained. These areas of pus were detected only by the radiograph, there being but slight local symptoms or signs. Into the roof of the mouth of this patient a dentist had inserted an elliptically curved bar of metal as large as the lead of a pencil, with possibly some idea of correcting a dental displacement, but with no correction of the pus focus. Subsequently this patient developed a cervical spondylitis, which cleared up shortly after draining the abscess cavities. One may even go farther and state that some of this mechanical work entraps pus which before such interference was draining into the mouth cavity, and thus at least lessening systemic absorption. Crown-work and filling are two common pieces of mechanical work which, if improperly done, may be a means of thus entrapping pus. A cavity in which a small quantity of pus is allowed to remain may harbor virulent organisms, which slowly extend the inflammation to the surrounding tissues, setting up large abscesses with active systemic absorption. Case IV illustrates the action of imperfect filling by producing irritation and inflammation (see case reports, Case IV). The radiograph in this case showed a shadow which was thought to be a pus area, but proved to be only thickening of the tissues. Cultures from all the suspected areas and from the root of the imperfectly filled tooth (which was drawn) were negative. There was, however, a positive fixation in the blood for three

out of twelve strains of *Streptococcus viridans*. Four months prior there had been an acute abscess of a tooth not connected with this imperfect filling, but severe enough to cause pain and swelling and to require radical treatment. No cultures were taken at the time of the operation, but the positive fixation found later and the inflamed areas about to become purulent were significant. This case seems to fall into a group of cases of latent streptococcic infection in which faulty dental work would be a sufficient exciting cause to establish an active local process, with systemic absorption. The faulty dental technic in crown, filling, and other mechanical work here referred to is well known to dentists, and it is mentioned not as an arraignment of the dental profession, but for the information of the general practitioner. It may be further stated that so far from being at fault, the dentists must be given the credit of pioneer work in dealing radically with pyogenic infection in and about the teeth and in the use of radiography in detecting such conditions. Evidence of this is found in the writings of such dentists as A. Witzel,² L. C. Ingersoll,³ W. J. Reese,⁴ Louis Jack, C. N. Pierce,⁵ and many others who published their observations between the years 1880 and 1900. While these were general considerations following the initial communication of John W. Riggs, of Hartford, Conn., which was made in October, 1875, they all showed an appreciation between these local dental conditions and systemic disturbances. The later writings of such dentists as M. I. Schamberg,⁶ formerly of Philadelphia; Harold S. Vaughan,⁷ of New York; Thomas L. Gilmer,⁸ of Chicago, to mention a few only, show an even greater appreciation of a probable relation between oral sepsis and many serious systemic diseases, an appreciation not yet shown by the majority of general practitioners. Among medical men, William Hunter,⁹ of London, and the late Dr. Kinnicutt,¹⁰ of New York, have been in recent years the most active in bringing before the medical profession the importance of this subject.

There are two chief factors militating against the thorough investigation and prompt treatment of these cases:

1. The dentist and the internist, through the improved standards of dental work, are coming together more and more in consultation and co-operation. This was, however, not always the case, and recalls the relation which existed between the surgeon and physician in England in the days of coffee-house consultation, in which the druggist was the middleman. The physician in those days merited his lower position in the scientific world, and the surgeon justly felt that these doctor druggists were not men worthy

²Vierteljahrsschrift f. Zahnheilkunde, 1882; British Jour. Den. Sci., xxv, 153.

³Ohio State Jour. Dent. Sci., 1881, 1, 189.

⁴Dental Cosmos, 1886, xxv, 550.

⁵International Journal, 1892, 1894 and 1895, xiii, xv and xvi.

⁶Dental Cosmos, January, 1906; Dental Brief, June, 1906.

⁷Jour. Allied Dent. Sci., March, 1913. (In this communication Vaughan shows the infection to be due to Vincent's bacteria without typical pus pockets.)

⁸Illinois Med. Jour., March, 1912.

⁹British Med. Jour., July, 1900.

¹⁰Proc. Roy. Soc. Med., April, 1913, vol. vi, Sup. No. 7, May, 1913.

to coöperate with. As the internist built up his diagnosis upon scientific research, it became possible for the surgeon to meet with him and study the case. Such is true also of the dental profession, and in 1904 the science had reached such a degree of accuracy that a section of dental surgery was established in the British Medical Association,¹¹ in which dentist and physician participate. Throughout the country to-day there is developing a class of dentists whose opinions are based upon carefully weighed evidence, and who are devising and carrying out operative measures as delicate in technic as some of the more important surgical operations. This first factor against the proper care of these cases can be best removed by the direct consultation between dentist and internist and their continued co-operation in the care of the case.

2. The second factor is the tenacity with which men and women alike will insist upon retaining rotten fangs in the mouth. The dentist regards the loss of a tooth as the internist does the death of a patient, and in this opinion the dentist has the insistent support of the patient. It is not an exaggeration to say that in saving certain teeth, the dentist may be condemning the patient to a living death through subsequent crippling arthritic infection or actual death through septic invasion of some vital organ. This factor can best be removed by educating the public, through the medium of the internist and dentist, to the realization of the seriousness of these conditions. When this is correctly understood by the patient, the removal of the offending teeth will be as readily consented to as tonsillotomy or the opening of a mastoid. Dr. Hunter¹² has dealt very thoroughly with the obligation of the dentist in this matter, and his opinions are warmly supported by many of the best dentists. I believe, however, that some of his remarks may be taken with great directness by the internist, who is not infrequently the first under whose observation these cases come.

REPORT OF CASES

CASE. I.—W. W., female, aged thirty-five years. Married. April, 1912, complained of pain in the left ear, for which she was referred to a specialist, who reported subacute inflammation of the left Eustachian tube. She was under treatment for this from time to time during the summer, but her condition not improving, she consulted, of her own accord, another specialist and was told by him that he found nothing wrong with the throat, ear, or nose, and by him was referred back to me. October 7, 1912, the patient seen again by me, when the following notes were made:

Complaint. Pain and tenderness located in the left ear. Distinctly audible crepitation of the left temporomaxillary joint.

Family History. Negative, except that one brother, aged forty-five years, is a victim of arthritis deformans, which began about nine years ago.

¹¹British Med. Jour., November 19, 1904.

¹²Practitioner, December, 1900.

He was acutely ill for several months, and while the joint condition was not progressive, he had occasional attacks of fever. He had been seen by several of the prominent consultants of this country. One sister (also under my care) has had septic infection of an antrum, for which she was treated by a specialist. Another sister had involvement of the small joints of the hands about the same time that the brother became infected.

Past History. Presented nothing of importance, except that wine was taken freely with meals and at other times also. The patient had one child. There was no history of miscarriages or pelvic infection.

Physical Examination. Presented nothing of importance that has not already been given. The patient was slender and of nervous temperament.

BLOOD EXAMINATION.

Hemoglobin (Fleischl).....	78 per cent.
Red cells.....	5,200,000
White cells.....	7,400

DIFFERENTIAL COUNT OF WHITE CELLS.

Polynuclear neutrophiles.....	78 per cent.
Transitional cells.....	0 per cent.
Large mononuclear cells.....	1 per cent.
Large lymphocytes.....	0 per cent.
Small lymphocytes.....	19 per cent.
Eosinophiles.....	2 per cent.
Mast cells.....	0 per cent.

100 per cent.

Urine examination was negative.

October 12, 1912, the patient was seen in consultation with Dr. Currie (dentist), and radiographs of the temporomaxillary joint and teeth were studied. Nothing was observed in the joint, but suspicion was directed to a tooth, which it was determined to remove. In the socket and about the pulp of this tooth pus was found, cultures from which were taken by Dr. Hastings at the time of its removal. Bacteriological report was as follows: Cultures from the tooth, the tissue, and from the tooth socket gave pure *Streptococcus viridans*, and cultures from pus about the gum and about the tooth gave *Streptococcus viridans* and *Staphylococcus aureus*. A blood culture taken a few days later showed no bacteremia, but for seven strains of streptococci the fixation was negative for four strains, and positive for three strains of *Streptococcus viridans*. There was no fixation for the gonococcus, and the blood for lues was negative. We had, therefore, evidences of a local *Streptococcus viridans* abscess, with systemic absorption. That the temporomaxillary involvement was a manifestation of this sepsis was a reasonable conclusion, borne out by subsequent developments. The cultures were preserved for the purpose of employing vaccines at a later date should the removal of this tooth and a cleansing of the socket not prove curative, or should other joints show evidences of infection. The patient continued under general observation, with marked improvement of

the ear condition. The crepitation of the temporomaxillary joint continued, but without pain or tenderness. In November, 1912, the patient went West, where she remained about two weeks. During this time she experienced, for the first time, pain in the ankles, knees, and wrists. There was no swelling, redness, or other local signs. On her return to New York, in the latter part of November, 1912, the pain continued in the joints noted, and extended to some of the finger-joints and to three toes of the right foot. The pain was worse in damp weather. This condition became sufficiently severe to alarm her, and on December 27 she reported to me. There was no fever and no local signs, except slight tenderness on moving the larger joints and on pressing some of the smaller joints. There was pain for the first time in the left temporomaxillary joint. There had been no chills or sweating. The knee-jerks were present. The pupils reacted normally, and there was no Romberg. It was decided to begin vaccine, and she received her first inoculation January 9, 1913. Up to April 21 she received twenty inoculations. By March 31, except for slight pain in a toe-joint of the right foot, the joints were free of all symptoms, a condition which up to that date had existed for about three weeks. She felt better than she had at any time for several years. The details of the vaccine therapy are omitted. By April 14, 1913, the patient complained of considerable tingling in the legs, and swelling and puffing of the feet. There was no fever, and though she had maintained her weight, and her appetite was good, she looked badly. April 21 she complained of feeling wretchedly; joints were more sensitive, and she noticed puffiness of the feet, especially at night. The diet and hygienic surroundings were carefully regulated and a tonic was prescribed and the vaccines were increased. She however continued to do badly. Blood examination showed the following:

BLOOD EXAMINATION.

Hemoglobin.....	100 per cent. (Sahli corrected)
Red-blood cells.....	4,720,000
White-blood cells.....	9,000

COLOR INDEX 1. DIFFERENTIAL COUNT.

Polynuclears.....	71.0 per cent. (300 counted)
Lymphocytes.....	19.6 per cent.
Large mononuclears.....	5.0 per cent.
Transitionals.....	3.6 per cent.
Eosinophiles.....	0.6 per cent.
Red-blood cells normal	

Blood negative for organisms, but the complement fixation was positive for the same strains of *Streptococcus viridans* as in the test made October, 1912.

On May 7 the teeth were again thoroughly radiographed, and though a careful study of these by two dentists and myself failed to detect pus shadows, Dr. Currie discovered a small area of pus near a devitalized tooth

adjoining the seat of original infection. There was also impingement of a neighboring tooth, which accounted possibly for the pain. This tooth was removed and the area thoroughly curetted, when rapid improvement followed. The local pain entirely disappeared, and in two or three weeks the joints gradually ceased to cause trouble. The general health improved and except for occasional observations by the dentist the case passed from medical attention. The crepitation in the left temporomaxillary joint persisted, and had been sufficiently marked to be audible to those sitting nearby. This joint, therefore, was probably permanently damaged.

CASE II.—W. R., male, aged forty-four years, married; seen May 27, 1913; complained of pain and tenderness in the vertebral column about opposite the seventh cervical.

Past History. Presented nothing of importance. Had no children, and wife had had no miscarriages. Used alcohol and tobacco in moderation. Gave an indefinite history of some trouble with the knee-joint, which he attributed to trauma two years before; occasionally felt slight twinges of pain in this knee on motion. Had had slight pain at times in some of the toe-joints. Was an active business man.

Present History. Vertebral trouble began about January, 1913; not progressive and not noticeable when sitting or lying still. Up to June, 1912, took recreation in boxing, but since then on account of the neck condition, had not been able to do so. At times he feared to move because of the pain. In every other particular his general health was good.

Examination. A large, well-built, healthy looking man; dark complexioned, of American-German parentage. There was no pigmentation of the mucous membrane, and the skin pigmentation was uniformly distributed. Carried his head in a fixed position, suggesting "stiff neck." On lightly tapping the top of the head tenderness was localized to about the sixth cervical vertebra, at which point the pain was localized; there was also here well-marked tenderness on pressure. A radiograph disclosed no definite change at this articulation.

BLOOD EXAMINATION.

Hemoglobin (Fleischl).....	85 per cent.
Red cells.....	6,016,000
White cells.....	6,000

DIFFERENTIAL COUNT.

Polynuclear.....	62 per cent.
Large lymphocytes.....	4 per cent.
Small lymphocytes.....	26 per cent.
Eosinophiles.....	3 per cent.
Transitional cells.....	5 per cent.
	100 per cent.

Radiographs of the teeth were studied with the dentist, Dr. Currie, and suspicion was directed to the upper right canine and the upper incisor

(see Figs. 2 and 6); in addition the dentist detected discharging pus from the upper right canine. On dealing with these teeth radically, pyorrhea, with marked destruction of the alveolus of the upper right canine, which was discharging, was found; also central necrosis of the right upper incisor, which, in Dr. Currie's opinion, was of some duration, together with a pus pocket in the lower left molar under an imperfectly fitting crown. Cultures by Dr. Mann gave *Streptococcus viridans*. The offending teeth were removed and all the infected areas thoroughly cleaned out. Tonic treatment was at the same time instituted. One week after this radical treatment the condition of the neck, which had existed for five months, markedly improved, and by the end of June entirely disappeared. This case had been under the observation of a dentist, who was consulted for the routine care of the teeth, and who entirely overlooked the purulent condition, inserting in the mouth the badly fitting crown and a bar for the correction of a suspected displacement.

CASE III.—J. F., male, aged forty-three, admitted to the City Hospital January 7, 1914. Laborer, widower.

Complaint. Pain in head—location indefinite; vomiting, chills and fever.

Past History. Venereal, denied infection. One year ago, while in Savannah, he had chills and fever which he says was "malaria." Alcohol in excess up to one year ago.

Present History. Chills, fever, vomiting, and headache for past four weeks.

Examination on Admission. Well nourished, looks sick, is apathetic, breath foul, tongue coated. Teeth: several missing, remaining ones covered with tartar; four lower front teeth move freely and can be lifted and lowered in their sockets; bloody white discharge oozes out of gums in moving these teeth; discharge is foul smelling; gums in general bleed on least touch, not tender.

Few palpable glands under chin; no glands elsewhere; throat negative; chest, abdomen and extremities negative; blood shows slight reduction in hemoglobin; no leucocytosis or oligocythemia. Blood culture negative; urine negative. January 16, 1914, submaxillary abscess; January 17, 1914, abscess opened and drained. Culture from abscess gives four different growths, which cannot be separated or identified. The temperature chart (see chart) shows the irregular fever which ran *while the infection was working down through the gum into the bone of the lower jaw* to point just below the chin. The radiograph here reproduced shows five lower front teeth, about four of which pus pockets can be seen (see Fig. 16). A radiograph which I regret has been mislaid but which I showed to the Stamford Medical Society presents a marked necrosis of the lower jaw bone in the region of the chin. This radiograph showed also the abscess sagging below the chin.

This patient absolutely refused to have the teeth removed. He illustrates well the ignorance amounting to stupidity which I am sorry to say

By this time a curable local condition will have become a systemic disease beyond even the possibility of the making of an autogenous vaccine. He then begins his rounds of our institutions as a case of "rheumatism," "arthritis," "gonorrheal joints," "arthritis deformans," "endocarditis," "chronic valvular disease," and he is studied with the aid of expensive laboratory equipment, radiograph, cardiograph, etc., and his joints are baked and the blood is cultured, and he is filled with salicylates and lectured upon, all of which is the study of ruins, the original fire having begun on January 7, 1914, when the small periodontal abscess was confined to that local process.

This case is full of instruction, but space does not allow of further analysis. I would like to add two points, however: First, other possible foci of pus tonsils, peritonsillar tissue, deep urethra, etc., must not be overlooked in searching for areas of infection; second, the general health of the individual often determines whether such infections are mastered by the defenses of the system or whether the infection spreads to other parts of the system.

CASE IV.—J. J., female, aged forty years; married. Seen September, 1911. Pain and tenderness in left knee-joint.

Past History. Nothing of importance. Borne two healthy children; no miscarriages. Had been in uncertain health for some years, but condition was attributed to nervousness, nothing definite manifesting itself.

Present History. The present trouble began with pain and tenderness on the outer aspect of the left knee-joint, aggravated by walking. This joint was treated locally by an orthopedist, and after eight months of wearing a brace and being under general tonic treatment, baths, etc., other joints became painful, including the temporomaxillary joints, making the chewing of food and articulation difficult. One year after the initial knee condition, fever of an irregular type appeared. Eighteen months from the onset an intermittent odor of the breath was noticed, and this first attracted attention to the teeth, which had given only such signs as were considered common and unimportant. On thorough examination of the mouth, pus was found to exude from the gum about a molar tooth, which, being removed, disclosed a pus cavity in the gum, from which was obtained a dram of pus. From this pure culture of *Streptococcus viridans* was obtained. The jawbone was found to be partly involved in the purulent process. The patient sustained a typhoid temperature for four months, with the involvement of many of the large and small joints. Autogenous vaccine was used continuously over a period of three years. During this time phlebitis in one leg developed. For ten months there has been steady improvement in the general condition, but several of the joints seem permanently damaged, and the knee-joints are fixed (by a possible tendon or muscle shortening) at an angle of 45 degrees. This case will be published elsewhere in detail from the bacteriological, vaccine, and arthritis viewpoints. For this reason the above brief outline only is given.

CASE V.—C. S., widow, aged forty-two years; complained of malaise, indigestion, and broken sleep.

Family History. Unimportant.

Past History. Showed repeated attacks of pharyngitis, and an acute attack of nephritis, without known cause, four years ago. Had not been robust for eight or ten years, but never confined to bed. Twenty-five years before, a prominent specialist diagnosed a tuberculous apex in the right lung. From this she seemed to have entirely recovered. Appendicitis with operation six years before. Two years before had amenorrhea for several months. Menses had been normal for over a year. Four months before, while traveling, developed a painful swelling over an upper left molar. This was treated by a dentist seen in the emergency, and he drained away a considerable amount of pus and directed her to see her dentist as soon as possible.

Present history dates from November 27, 1912, with complaint of pus about a molar four months before, as given above, but there is no trouble with the teeth at present. Patient was always an active woman, attending to her own affairs, but is now unable to go about without prostrating fatigue. Appetite is poor, and she sleeps but a few hours at night. For the first time in her life she finds relief in remaining in bed.

Examination. Skin pale, but mucous membrane of fairly good color. Chest negative except for soft systolic blow over the heart. Abdomen negative. Over both lower extremities were pinhead, purpuric spots. There had at no time been any joint pain or tenderness.

BLOOD EXAMINATION, DECEMBER 2, 1912.

Hemoglobin.....	90 per cent.
Red-blood corpuscles.....	5,600,000

DIFFERENTIAL COUNT

Polymorphonuclears.....	61 per cent.
Transitionals.....	5 per cent.
Lymphocytes.....	24 per cent.
Large mononuclears.....	7 per cent.
Eosinophiles.....	3 per cent.
	100 per cent.

Urine Examination. Reaction, acid; sugar, none; pus, none; albumin, faintest possible trace; urea, 1 per cent.; casts, none; specific gravity, 1.018.

Blood Culture. Negative for organisms (agar and broth): for twelve strains of streptococci, the fixation was positive for three.

Examination of the Teeth. Pyorrhea pocket (see Figs. 2, 3, and 4) about a twelve-year molar. At the root of a filled tooth the radiograph showed a shadow, thought to be due to pus. Tooth was removed and filling was found to be incomplete, and from this inflammation extended into the surrounding tissues. Cultures from this area were negative. The shadow

in the radiograph was due to thickening of the bone and peridental membrane, secondary to the inflammation. Though the cultures from the tooth were negative, the blood showed positive fixation for three out of twelve strains of *Streptococcus viridans*, and had this local condition remained untreated, an abscess would undoubtedly have developed, as was the case with the other side of the mouth four months previous.

This and Case II perhaps represent an early stage of a local streptococcic infection with systemic manifestations. In both cases the systemic manifestations rapidly disappeared after thorough local treatment. Such cases require careful watching for the possible development of new areas of infection.

CASE VI.¹³—R. S., female, aged thirty years, unmarried; seen February, 1913, complaining of severe pain localized to the left tonsillar region and left ear. The pain radiates toward the mastoid and the neck posterior to the sternocleido muscle, with a center apparently in the external auditory canal. A thorough examination of the throat and ear by a specialist revealed nothing. There was no fever. The teeth were found to be exceptionally good. There was, however, tenderness on slight pressure on the gum below and posterior to the lower left third molar; the gum was slightly red and puffy. Conditions suggested an abscess, but the radiograph revealed a wisdom tooth impinging directly upon the molar and crowding that tooth (see Fig. 1). This condition, known in dentistry as "impaction," is a frequent cause of reflex pain, and without the use of the radiograph leads to much surmise with regard to possible mastoid and middle-ear disease, retro-tonsillar, deep gum, and dental abscesses. These cases are frequently treated by lancing the gum in the hope of liberating supposedly entrapped pus. The loss of sleep on account of the pain and disturbed digestion not infrequently lead to the impression of a systemic condition secondary to the supposed abscess. This case was surgically treated by Dr. Schamberg, who removed the offending tooth, after which the patient was entirely relieved.

SUMMARY. These cases are selected as representing six important features of general disturbance of the system, secondary to dental disease. In the first case, the dental disease was detected in part and the organism determined before the joint complications developed. The use of autogenous vaccine in this case would seem to have held the progress of the disease in check, but not until all of the pus areas in the teeth had been cleaned out did the systemic condition show a permanent improvement. The system, however, was probably under the influence of toxic absorption for some months before radical measures were instituted. In such cases there is always a possibility of new and independent foci of infection having been set up. Such a stage in the development of the disease precludes the possibility of checking it by dealing with the original focus merely, and it would

¹³This case represents a class well-known to dentists, but one with which general practitioners are usually unfamiliar. It is introduced here, however, for the information of the general practitioner in order that he may distinguish it from beginning dental sepsis, with which it can be readily confused.

seem here that vaccines are our only means of combating the infection. This is true of Case III, in which neither the organism was detected nor the autogenous vaccine employed until many months after the system had been under the influence of the infection and many joints involved, but this case also represents the ravages of septic infection of dental origin unchecked, as was common with all cases before the teeth were suspected as the original focus. Cases II and III represent possibly the earliest stage in the systemic manifestation of absorption of toxins from a dental source. Case VI represents a class in which mechanical disturbance may be mistaken for an inflammatory process. From these cases and others the following symptoms and signs may be classified. In attempting a classification, it should be stated that cases of this type have not, as yet, been studied by internists and dentists together sufficiently to make such a classification complete. It should also be stated that there are a number of characteristics which may prove of great clinical value when corroborated by thorough observation, but which for lack of such corroboration are omitted here. Such, for example, are the association of joint involvements in which the upper jaw is the seat of pyogenic infection in contradistinction to an adenitis and burrowing pus when the lower jaw is involved. Again, what degree of alveolar destruction is necessary before systemic absorption takes place? Also, is there any relation between tonsillar and peritonsillar and faucial infections and dental disease? In this connection, Dr. Vaughan's¹⁴ observations with regard to Vincent's bacteria and dental disease are pertinent. We are as yet without any conclusive studies on the lymphatic system draining such areas.

SYMPTOMS. Malaise, indigestion, fetor of the breath, often intermittent; there may be periods in which the breath is so offensive as to be noticeable some little distance from the patient, while at other times such fetor cannot be detected by careful search. This is frequently called attention to by others, and is rarely noticed by the patient. Loosening of the teeth, which may also be intermittent. Tenderness of the teeth on chewing; an indefinable aching of the gums; pain referred to the ear (internal and external), the mastoid, the cheek bone, the eyes, and fauces. Rapid and sometimes copious loss of hair, which may occur intermittently. All the frank manifestations, such as acute pain and swelling, which immediately attract attention to the teeth, are omitted here, though of course they come into this classification.

SIGNS. A brick-red tinge about a sixteenth of an inch in width at the dentogingival margin, quite distinct from the whitish-red color of the healthy gum, marks areas of infection. These areas bleed readily on slight pressure. If a wooden spatula is run along the gum margin, the diseased portions can frequently be detected by the bleeding-points left in the wake of such pressure. In more advanced cases, a diseased tooth, from loosening, can be moved in its socket; a healthy tooth is absolutely immobile. Again, the gross signs, such as destruction of the cemental portions, surface necroses, and visible discharge of pus, together with more obscure signs requiring

¹⁴H. S. Vaughan, Jour. Allied Dent. Soc., December 2, 1912.

instrumental examination by a dentist, are not mentioned here in detail, though they of course belong under this head. The most important point in connection with both symptoms and signs is that manifestations of systemic involvement may appear without there being any symptom referable to the teeth in which actively absorbing pus areas may be present. Such systemic manifestations, however, show marked improvement, and sometimes com-

FIG. 1



FIG. 2



FIG. 1.—Normal teeth, for comparison. The roots will be seen to blend with the gum, showing no areas between the teeth and gum. The gum fits snugly up to the teeth, all the way up and between the roots. There is, however, in this case an impacted molar which can be seen (to the left of the picture) impinging on its neighbor. This condition may give reflex pain in the ear, neck, etc., and may be mistaken for pus formation (see Case VI). It is a favorable location for the development of pus.

FIG. 2.¹⁵—Simple pyorrhea pockets, early stage (left lower first molar).

FIG. 3

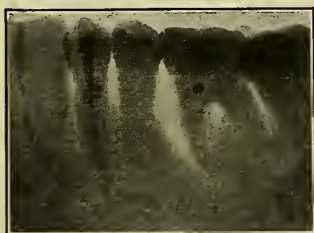


FIG. 4



FIG. 3.¹⁵—Simple pyorrhea pockets; more advanced stage; no involvement elsewhere.

FIG. 4.¹⁵—Typical pyorrhea alveolaris; no involvement of pulp.

pletely disappear with the radical removal of the septic focus. Paramount among these systemic manifestations are arthritis and phlebitis. It is a noteworthy fact, however, that not until an overwhelming sepsis has developed does one procure cultures of any organism from the blood or the joint fluid. A positive complement deviation, on the other hand, from the blood is a fairly uniform finding. Fever, except in advanced cases of systemic involvement, is not common. Grave anemia of oligochromemic type may be present in the early stage of dental sepsis. Oligocythemia occurs in cases in which the system has been involved for some time. Grave secondary anemia may

¹⁵If the general health is not taxed by bad food and bad air, overwork, worry, etc., the condition shown in Figs. 2, 3 and 4 may exist for long periods with or without local manifestation, but usually with moderate systemic disturbance (see Case IV). With the extension of the local process to the tooth or surrounding tissues or with deterioration in health from any cause, the system may become suddenly and seriously involved (see Case II).

develop in such cases. Polynuclear leukocytosis or increase in the number of the white cells is rarely found, even when large pus areas exist in and about the teeth. Glandular enlargement seems to occur more commonly

FIG. 5



FIG. 6

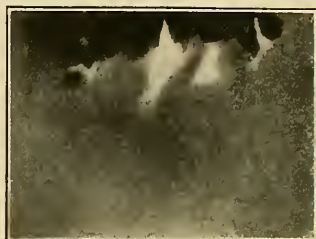


FIG. 5.—Pus in a bifurcation, discharging into the mouth. Destruction of tooth and systemic absorption imminent.

FIG. 6.—Pus pockets with necrosed roots under crowns, systemic absorption active. (See Case II.)

FIG. 7

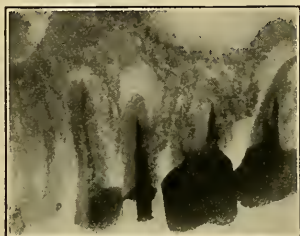


FIG. 8

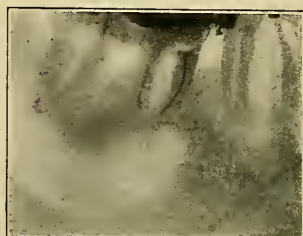


FIG. 7.—Abscesses over bicuspid roots. Advanced stage. Systemic absorption active.

FIG. 8.—Large abscess cavity in lower jaw; considerable loss of bone through septic process.

FIG. 9



FIG. 10



FIG. 9.—Chronic abscess; tooth structure entirely destroyed; tooth floating. No local symptoms, except mobility of tooth and sinking of tooth when patient chewed.

FIG. 10.—Absorption of root by abscess.

with lower than with upper-jaw involvement. The submental and cervical glands are those usually affected.

DIFFERENTIAL DIAGNOSIS. While the occurrence of pus in and about the teeth has been found to be the cause of serious sepsis in various parts of

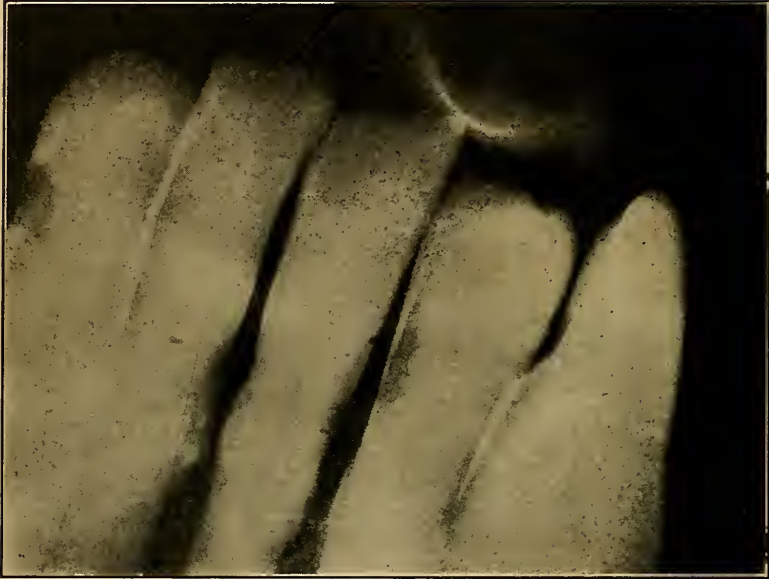


FIG. 17.—Enlarged skiagram of teeth with pus pockets as seen in Fig 16.

FIG. 11

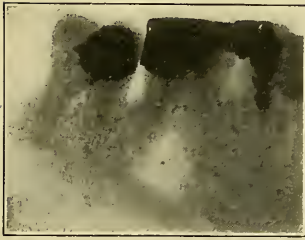


FIG. 12



FIG. 11.—Advanced abscess; no signs or symptoms, detected by radiograph only.
FIG. 12.—Chronic abscess.

FIG. 13



FIG. 14



FIG. 13.—Combined Riggs' disease and dental abscess; chronic abscess with fistula; pulp extensively involved.

FIG. 14.—Perforated root; considerable loss of bone.

FIG. 15

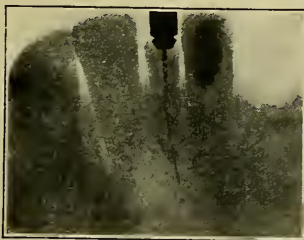


FIG. 16.

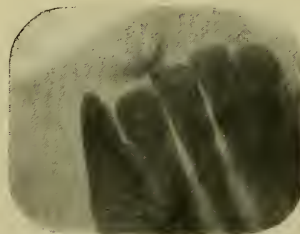


FIG. 15.—Chronic abscess; lateral root; pus draining through canal of pulp; absorption until pus was liberated.

FIG. 16.—Four lower front teeth with pockets of pus which drained into lower jaw bone and pointed below the chin.

the system in a large number of cases, the search for primary foci of pus other than the teeth must be thorough. The deep urethra, the tonsils, the peritonsillar tissues, the antrum, the posterior nares, must all be excluded. The testing out of the blood for complement deviation for various organisms may prove the most direct evidence in the absence of local manifesta-

tion. It may be well to repeat that the blood gives negative cultures unless an advanced degree of sepsis has developed, and in such cases the condition has usually gone beyond the stage in which it can be influenced either by local, general, or vaccine treatment. Case III, while the condition remained localized to one knee, was suspected of being tuberculous. Its real character was detected only when pure cultures of *Streptococcus viridans* from the tooth abscess and a positive complement fixation of the blood for the same organism were obtained, the blood otherwise being negative. Fluid from the knee-joint was also negative.

Radiographs of the teeth should be placed first among the means of detecting pathological conditions in the jaw. In some instances, where the pus lies in a thin layer over the tooth and not in sufficient quantity to produce a pocket, nothing may appear on the plate. This occurred in Case I, when by a second examination the pus was reached by probing only.

Reference has already been made to impaction and misplacement, with the consequent swelling and reflex pain which may simulate these septic conditions.

TREATMENT. A realization that one is dealing with abscess cavities, due possibly to the most virulent organisms, with the inevitable absorption of destructive toxins, is essential to the prompt and radical cure of these cases. While it is true that the teeth (and possibly the tissues about them) resist the entry of organisms themselves into the system, the possibility of a bacteriemia always threatens such cases, with probably the development of new foci of pus in the endocardium and other like vital organs. Abscesses in and about the teeth should be attacked with the same energy as pus foci in any other part of the body. Infected teeth should either be removed entire or in part by operating above the tooth through the gum, an operation now done with great skill by many dental surgeons. Cultures should always be taken at *the time of the removal of the tooth or the operation*, and in the event of a positive culture, vaccines should be prepared and stocked in case they may be needed at some later date. Filling of the teeth and temporizing with a hope of saving a tooth which has been attacked by pus should only be permitted after careful consultation with a competent dentist. The physician is under obligation to the patient to make him realize the seriousness of the possible consequences of the seemingly unimportant condition of the teeth. This part of the treatment cannot be too strongly emphasized, and the responsibility of temporizing with or retaining septic teeth should be placed by the physician and dentist upon the patient.

Through the courtesy of Drs. Vaughan and Tracy, already mentioned, and by the aid of the City Hospital X-ray Department, I have the opportunity of showing radiographs illustrating the cases reported here as well as the location and distribution of pus in and about the teeth and gums.

CLOSURE OF THE ABDOMINAL INCISION

BY

CHARLES G. CHILD, JR., M.D.,
ATTENDING GYNECOLOGIST

CLOSURE OF THE ABDOMINAL INCISION

BY CHARLES G. CHILD, JR., M.D.,
Attending Gynecologist

In the closure of every abdominal incision, one immediate indication must be met—the prevention of primary hernia. Does the almost invariable custom of closing the wound with absorbable suture material meet this indication? To my mind, it does not; and I believe that it leaves much to be desired along other lines as well. In the early days of abdominal surgery, when speed was a factor that had always to be reckoned with, and before the introduction of absorbable suture material, primary hernia was extremely rare. This was because the wounds were closed with heavy silk sutures. While the primary results, so far as hernia was concerned, were good, the late results were poor because of the low percentage of primary union that was obtained in those days. When, later, rapid operating became of less importance, more attention was given to closing the wound. With the introduction of absorbable suture material, closure by approximating layer to layer in proper anatomical relations was adopted. This, probably because of improved asepsis, gave a higher percentage of primary union, with less frequent occurrence of secondary hernia in cases clean at time of operation. In cases septic at time of operation, however, this method yielded a low percentage of primary unions, very much lower than in clean cases. With the further improvements in technique of McBurney and Pfannenstiel, post-operative hernia, both primary and secondary, have been practically abolished, and to-day the primary consideration in closing an abdominal incision made by either of these methods is to secure primary union, and thus avoid the prolonged convalescence that follows in the wake of an infected wound. In the case of the median-line abdominal incision, still so often employed, much greater care has to be used in suturing, both as regards the suture material and the method used, for in such a wound far greater strain is brought to bear upon the sutures uniting it. Just how often a median-line incision in the lower abdomen, closed with absorbable material, blows open shortly after operation, it is difficult to say. Certainly the accident happens far more frequently than it should.

To paraphrase a well-known saying: "Surgery is much like eating: we cannot always tell what did the patient good, but we can usually tell what did him harm."

When an abdominal wound breaks open during a violent fit of vomiting, it is fair to presume that the sutures used to unite it were not strong enough. Likewise, when a low percentage of primary wound union obtaining with

one suture material immediately yields to a high percentage when another material is employed, it is fair to presume that the first was at fault.

What, then, are the requirements we should strive to meet in closing every abdominal incision? It seems to me that two are of paramount importance—prevention of hernia and the securing of primary union.

Hernia.—When this occurs shortly after the operation, it is always due to a sudden severe strain. Coughing or vomiting are the most frequent causes of such an accident. Investigation into these cases will usually show that the material employed was of insufficient strength, or had been weakened by premature absorption in the tissues. Catgut has probably been most at fault in this respect.

During the past year I have listened to reports of six cases where this accident occurred. In all, the wound had been closed with catgut, and without exception the secondary suturing was done with silkworm gut. Secondary post-operative hernia, when not caused by defective technique in closing the wound, is due to infection of the wound. In this respect, it is interesting to hear the opinions of several surgeons of wide experience. Von Rosthorn writes: "During five years I practically saw no ventral hernia in my cases, except when suppuration had taken place." Bier: "I think it depends less upon the suture material used, and the so-called 'method,' than upon a satisfactory healing by first intention. If this does not succeed, then other measures fall short. I hardly ever remember seeing a post-operative hernia when healing by first intention has strictly occurred." Von Eiselsberg: "I have never found a hernia, provided that healing took place by first intention." Dührssen: "When, however, an abscess occurs in the wound, a hernia follows, no matter how the wound was originally sutured."

It is of the greatest importance in closing abdominal wounds that the tissues should be left in their proper anatomical relations, and so retained, without undue tension or constriction, until permanent union has taken place. When infection occurs in a wound closed with absorbable suture material, these sutures become likewise infected, and slough out, releasing the tissues, so that secondary suturing is necessary if the patient is not to be left with a permanently weakened abdominal wall.

The abdominal incision differs from incisions in all other parts of the body in one very important point—and this is particularly true of the lower abdomen: it is frequently subjected to great increase in tension. This fact should always be taken into consideration when closing the wound, especially in closing one made in the median line, where the strong outward pull of the oblique and transversalis muscles causes an almost constant strain on the sutures. This strain is greatly increased when retching or vomiting occurs; therefore the necessity of suture material of known tensile strength that will not be weakened by premature absorption or infection before the fascia is firmly united.

Separation of the fascial edges, with a high percentage of post-operative

hernia, is the rule in wound union by secondary intention. We often hear the remark, "It makes no difference how the wound is closed, provided primary union is obtained"; hence the necessity of a method of closing the incision that will give the highest percentage of primary union.

Of the many features entering into the process of wound infection, two I believe to be of paramount importance: undue traumatism of the tissues at the time of operation, and the use of absorbable suture material in septic cases. Traumatism greatly lessens the vitality of the tissues and their power of resistance, while absorbable suture material introduces what I believe to be an excellent culture medium. Before absorption of the suture material can take place, it must be converted into a soluble gelatin. Gelatin is an excellent culture medium upon which bacteria readily grow. The tissues, when in a normal or approximately normal state, will readily take care of a small amount of infection if the wound has been closed with noninfectable suture material, and is free from areas of necrotic tissue. It is a different story, however, in a wound containing many areas of traumatized tissue, and where numerous foci of pressure necrosis of the tissues has been produced by the tying of strangulating sutures. Such a wound falls an easy prey to subsequent infection, as most anyone's clinical experience will show, and yields a very low percentage of primary union.

Halsted, in an excellent article on ligature and suture material recently published in the *Journal of the American Medical Association*, makes the following pertinent remark: "Catgut, even that which we have no cause to believe is not sterilized, irritates the wound for some reason, perhaps because it serves as culture medium for saprophytic organisms which are carried into it from the top epithelium and follicles of the skin. We have frequently observed this reaction, and have occasionally had an opportunity to compare the reaction caused by catgut with that of fine silk in wounds clinically studied in the same patient at the same time. Let the surgeon interested in making the comparison, when he has occasion to amputate both breasts for malignant disease, take a running subcuticular stitch on one side with catgut and on the other with fine silkworm, and observe the healing wounds from day to day; or when operating on two goiters employ catgut or platysma suture in the one case and very fine silk in the other. There is not only greater local reaction in the cases sewed with catgut, but in them the wounds will occasionally open at one or more points to discharge a few drops of clear or cloudy fluid."

This favorable difference is even more marked with silkworm gut than with fine silk, and where silver wire is used no evidence of any local inflammatory reaction can be observed. Why, then, continue to produce in the wound conditions so liable to defeat the very object we should strive most to obtain? Prejudice has a great deal to do with this question. As a good illustration of this is an answer I once received from a prominent surgeon, to whom I put the question: "Why do you use absorbable suture material of unreliable strength and uncertain absorbability?" His reply was: "I use

suture material that is absorbable because my patients object to the removal of nonabsorbable sutures more than they do to the operation itself." More, I wonder, than they would to the dangers of primary hernia or to a prolonged convalescence accompanying an infected granulating wound, with a possible secondary operation for hernia.

I have never believed that absorbable suture material should be used in the closure of the abdominal incision, for its unreliability is too great. In cases septic at time of operation, I feel certain it should never be used.

Only a short time ago, in discussing this point with a surgeon who had just finished reading a paper on a new method of wound closure with catgut, I called his attention to the fact that his cases showed only about 67 per cent. primary unions. "Oh," he replied, "but many of the cases in this series were suppurative processes, where it is always difficult to get primary union!" This seems to be a very current surgical belief; that it is a heresy I shall endeavor to show.

Some years ago I published my results with the use of catgut and with silk as a suture material in closing the abdominal incision. The results were as follows:

CATGUT

Primary union in clean cases.....	70.5 per cent.
Primary union in septic cases.....	50.0 per cent.
Percentage of cases septic at time of operation.....	26.0 per cent.
Average primary union, 60.2 per cent.	

The catgut not only gave a low percentage of primary union in the clean cases, but in the suppurative processes actually yielded one infection for every two cases operated upon. I then abandoned this material, and in the next series of cases used buried silk and silkworm gut in the skin with the following gratifying results:

Primary union in clean cases.....	91.2 per cent.
Primary union in septic cases.....	95. per cent.
Percentage of cases septic at time of operation.....	29.8 per cent.
Average primary union, 93.1 per cent.	

Since that time I have used a method which I described in the *Journal of the American Medical Association* in 1907, and with which the results have been so near perfection that I feel it worthy of a more extended report. The essential feature of this method, closure of the wound with nonabsorbable sutures fastened outside of the skin and subsequently withdrawn, I afterward learned was described by Dr. Haughey of Battle Creek some years before my article appeared, and to him I gladly acknowledge priority. We both used silkworm gut, his being an over-and-over stitch shotted outside the wound, while mine was a continuous mattress stitch, the ends tied over gauze. The mattress stitch I found gave better approximation of the tissues, and could be more easily withdrawn. The long ends permitted a reopening of the wound when necessary without taking out the sutures.

This method will guard absolutely against tearing open of the wound, and will give a high percentage of primary unions. In the hands of a clean

operator, it will give primary union just as often in the cases septic at time of operation as in the clean cases. I now use a No. 23 silver wire introduced on a linen thread carrier in place of silkworm gut.

TECHNIQUE.—The closure of the wound is effected as follows:

Peritoneum.—Where the opening is small, it is closed with a purse-string suture of fine silk or linen thread. This introduces a minimum amount of suture material, and turns the raw edges of the peritoneum outside of the abdominal cavity, preventing intestinal or omental adhesions. Longer incisions are closed with a continuous suture of silver wire brought out through the skin, or a buried one of fine kangaroo tendon.

Fascia.—Here a continuous mattress suture is used. The free ends are brought out through the skin, one inch from the angles of the wound, and fastened over a roll of gauze to the free ends of the subcuticular stitch uniting the skin.

Fat.—When the fat is of excessive thickness, it is likewise brought together with a continuous suture, emerging at the angles of the wound.

Skin.—A subcuticular suture brings the skin together. The free ends are brought out through the skin one inch from the angles of the incision and on the opposite side from the ends of the fascial stitch. A small roll of gauze is then laid over the wound, the sutures given a final tightening, and fastened by twisting, skin ends to fascial ends.

Subsequent Care.—On the second day, the dressing is taken down and the roll of gauze cut in two so as to allow proper inspection of the wound. This should be examined every day so that any effusion of serum or blood clots in the wound may be detected early and liberated before the formation of pus takes place. By this precaution wound infection from the suppuration of an unabsorbed effusion of blood clot will often be avoided. The more or less common practice of not looking at the wound for nine or ten days, or until an elevation of temperature and pulse develops, is only too often responsible for cases of secondary union. When the elevation of temperature and pulse occurs, pus has formed and secondary union is inevitable.

If infection should occur in a wound closed by the above method, the sutures are unfastened and the skin one withdrawn to allow free drainage. Where the infection extends under the fascia, the fascial suture is not removed, but the edges of the fascia are separated upon it to allow proper cleansing. Later, when the infection has subsided and union begun, traction on the free ends of the suture brings the fascia together. As this suture is not tied in the fascia, it does not slough out, but gives perfect control of the fascial flaps until removed. Thus, firm union of the fascia in infected cases as well as in clean cases becomes possible without resort to secondary suturing. If the case is one of appendicitis, where drainage has been employed, the free ends of the sutures are left loose until the drain is removed, and are then tightened.

By this method many indications are met. Halsted, in the article

above referred to, speaking of faulty technique, says: "One should not bring parts together in such degree of tension as to cause necrosis, or interfere greatly with the blood supply, for nothing is gained by so doing, and decided harm may result." To this we heartily subscribe. Every ligature or suture tied in the wound, if tied tight enough to accomplish its purpose, includes in its bite a portion of devitalized tissue, the center of an area of pressure necrosis, a foreign body, a menace to wound union; therefore, with our method, we tie no sutures in the wound. The ends of the sutures are brought out through the skin and fastened over a bolster of gauze outside of the wound. This method snugly approximates the tissues, but does not impair their nutrition or in any way interfere with their circulation. Furthermore, if at any subsequent time we desire to liberate the tissues temporarily, it is only necessary to release the outside fastenings.

I have spoken of the advisability of this in infected wounds, and would further call attention to its value in reopening the abdomen where secondary hemorrhage may have occurred.

In one of my cases, where this accident happened, the wound was reopened, the hemorrhage controlled, and the wound reclosed without the removal or reinduction of a single suture.

Dr. Halsted further says: "For sewing up an abdominal wound, where it is necessary here and there to take heavy top stitches, perforating skin and muscles, silver wire serves admirably; when the section can be approximated without tension, we usually employ unrepeatd perforating stitches of very fine silk. If the skin edges have to be brought together under considerable tension we recommend a subcuticular stitch of silver wire."

Seldom, if ever, in our experience have we found it necessary to take such deep mass sutures, and we notice with many operators a custom of reinforcing their abdominal wounds with such sutures, either of heavy wire or of silkworm gut. Some place as many as four or five of these through-and-through sutures in addition to continuous or interrupted sutures of catgut in the muscles, fascia, and skin.

This certainly is a complicated and time-consuming procedure. Of the tissues held by the through-and-through nonabsorbable sutures, the fascia is the only one of importance, and this, in our method, is held in closer and firmer approximation with one strand of silver wire than with numerous interrupted sutures enclosing the skin and muscles as well.

This "three-suture" method, which is also a "three-layer" method, appeals to me greatly, and the results obtained with it in a large number of cases have been most satisfactory. In the last 100 cases in the Gynecological Division, where it was used, the percentage of primary union was 95 and the percentage of cases septic at time of operation 17. In the five cases that failed to unite by primary union, the colon bacillus was responsible for two, a retained blood clot for two, and in the other a slight infection (the patient removed the dressing on the second day after operation, and tried to take the sutures out).

INTRAVAGINAL MYOMATA OF UTERINE ORIGIN

BY

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Attending Gynecologist

The following cases are recorded because of the rareness of this condition and the interesting clinical symptoms with which they were associated.

Intravaginal myomata of cervical origin are of much less frequency than are intravaginal myomata of uterine origin. The average of the former has been variously estimated at from 3 to 8 per cent. In my own series of one hundred cases, vaginal myomata were encountered in 1 per cent. and the intravaginal variety were met with in the same proportion.

Notwithstanding the rareness of this lesion, the ease with which a diagnosis is to be made would seem to almost prohibit the frequent errors with which it is associated.

Mrs. M. R., æt. 40 years. Was admitted to the City Hospital in June, 1911. Menstruation had always been regular, of four days' duration, profuse in amount, with slight pain. She had given birth to two children, the last one fifteen years previous; two miscarriages, in rapid succession, had occurred five years later. Severe pelvic pain and metrorrhagia were the only symptoms complained of. Examination showed an intravaginal myoma 5 inches in diameter. There was a slight vaginal discharge serosanguineous in character. The cervix could not be felt, nor could any fundus be made out above the symphysis; on the contrary, the abdominal wall being quite thin and relaxed, a cuplike depression where the fundus should have been located was quite easily made out. A diagnosis of intravaginal myoma of uterine origin with probable inversion of the uterus was made.

At operation on January 27th the tumor was removed by vaginal morcellation.

It was found to grow from the uterine wall near the fundus. During its protrusion through the cervical canal, it had drawn the small fundus with it, completely inverting the uterus as far as the internal os. The fundus was removed as well as the tumor and the cervix at the internal os sutured with one small strip of gauze for drainage.

Convalescence was uneventful.

Mrs. M. M. æt. 43 years. Was admitted to the City Hospital in June, 1912. She complained of severe pain and a sense of great weight and dragging in the pelvis. This condition had become progressively worse for the past two years. Accompanying this pain, there had been frequent and painful urination. Eighteen months previous to her admission to the Hos-

pital, micturition had become so difficult that the catheter had to be used once or twice during the twenty-four hours to thoroughly empty the bladder, and had been used constantly for the past eight months. Catheterization was performed with a soft rubber catheter which the patient carried in her pocket and which had never been cleaned other than rinsing with water during all of this time.

Menstruation was regular, seven days' duration, moderate in amount, and painless. Seven children had been born by normal deliveries, the last one eight years ago. One miscarriage at three months followed three years later.

The patient was sent to the Hospital with a diagnosis of inoperable carcinoma of the uterus. On examination, the patient was seen to have a profuse, purulent, fetid vaginal discharge, streaked at times with blood. The perineum showed signs of an old laceration with considerable relaxation of the vaginal outlet. The vagina was much distended and entirely filled with a large hard tumor presenting at the vaginal outlet. Several very much softened areas of necrosis were present in different parts of this tumor. The urethra was tightly compressed against the symphysis pubis by the tumor, and it was with difficulty that the finger could be passed between. It was impossible to feel the cervix per vaginam, or to map out the uterus above the symphysis. The diagnosis of fibromyomata undergoing necrosis, probably of cervical origin, was made.

At operation on June 13th the tumor was removed by morcellation performed by the vaginal route. The tumor showed many deep-seated areas of necrosis, as well as those which had been observed on the surface, and was about the size of a child's head at term. It grew from the posterior cervical lip. A thorough pelvic examination made after removal showed a small hypertrophic uterus and failed to disclose any disease of the adnexa.

Convalescence was uneventful. The patient voided normally five hours after operation, and at regular intervals thereafter. The urine, which before operation had been cloudy, with a specific gravity of 1.013, containing numerous blood cells and a few pus cells, was normal at the end of two weeks.

Of especial interest in this case is the fact that for a period of nearly a year self-catheterization was performed with a catheter which was never sterilized, and yet only a slight degree of cystitis developed, which very promptly cured itself within two weeks after the catheter life was stopped.

PRIMARY SARCOMA OF THE UTERUS—CASE REPORT

BY

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PRIMARY SARCOMA OF THE UTERUS—CASE REPORT

BY HENRY V. HOLCOMB, M.D.,
Assistant Attending Gynecologist

Sarcoma of the uterus, while comparatively rare, occurs with sufficient frequency to make it a matter of importance; and in all cases of uterine hemorrhage, of obscure origin, this condition should be borne in mind. It may occur at any age, but most of the cases reported have been shortly before or after the menopause. Statistics bearing on the frequency of this condition are confusing.

In 403 cases of malignant growth of the uterus reported from Halle, 387 were carcinoma and only 15 sarcoma. Gessner in a series of 9,133 cases of uterine tumors, both benign and malignant, finds 18 cases of sarcoma, while Roger Williams finds only 2 cases of sarcoma in 2,649 of uterine tumors.

The frequency of sarcoma as against that of fibroma is found by Martin to be 3 of sarcoma to 100 of fibroma; Kelly, 1 to 100; and Piquand, 2 to 100. In 1911, M. P. Bégouin reported 7 cases on which he had operated since 1903, and during that time he saw 130 cases of fibroma, making a ratio of 5 to 100.

The most frequent location is in the body of the uterus, though it also occurs in the cervix and in the vaginal portion. Sarcoma of the body of the uterus may be circumscribed or diffuse.

The clinical picture of the circumscribed variety in its early development closely resembles that of fibroma.

Many competent observers are of the opinion that this condition is often sarcomatous degeneration of a preëxisting fibroid, while others believe that these tumors are sarcomata from the beginning. However, the possibility of the presence or development of this condition is, in my opinion, a further indication for the early surgical treatment of fibromyomata.

The symptoms of the diffuse form of sarcoma of the body of the uterus are the same as those of carcinoma, and these two conditions can only be differentiated by the microscope.

In both varieties, hemorrhage is perhaps the most constant symptom, while pain is often absent or only very slight until the disease is well advanced. Of course, as the disease develops, the symptoms become much more marked, and are those of a malignant uterine growth. They are pain, cachexia, offensive watery discharge, and the presence of a rapidly growing tumor which soon becomes fixed and infiltrates the surrounding

tissues. Hemorrhage is now often profuse. Metastatic deposits in the viscera, especially the lungs, are frequent.

Early diagnosis can only be made by the microscopical examination of tissue removed for this purpose.

The prognosis is of course very bad, but when the growth can be entirely removed, the prognosis is perhaps better than in carcinoma as the lymphatic involvement in sarcoma is usually very slow.

The following case is one of diffuse sarcoma of the body of the uterus in a very early stage. This case illustrates very well the extreme importance of carefully preserving for examination all material removed by the curette.

MRS. F., age, 47; widowed; native of Austria; admitted to the City Hospital June 10, 1913.

Family History.—Negative.

She has had three full-term pregnancies. The first two were still-born, and the last one—ten years ago—resulted in a healthy, living child. Shortly after the birth of this child, she had a severe uterine hemorrhage, which was checked by a uterine packing. She has also had three miscarriages. The last one, three years ago, occurred at three months. The cause was unknown, and convalescence was uneventful.

Ten days before admission, the patient noticed a rather sudden flow of bright red blood from the vagina. This flow became quite profuse, and was accompanied by slight pelvic pain and a feeling of general weakness. She received medical treatment from her physician, but this condition persisted and grew rapidly worse.

Examination on Admission.—The patient is well-nourished, but presents the picture of a well-marked anemia. Examination of the chest and abdomen is negative. Vaginal examination reveals a freely movable uterus of moderate uniform enlargement. The cervix is rather soft, and from it appears a rather profuse flow of bright red blood. No further abnormalities were discovered in the pelvis.

Treatment.—The patient was treated by the usual palliative measures, but as the flow persisted with only a slight diminution, on June 18th she was given a general anesthetic. Examination under the anesthetic revealed nothing more than had already been learned. The cervix was very soft and dilated very readily. On introducing the curette into the uterus, a very profuse bleeding occurred and persisted until the uterus was tightly packed. The interior of the uterus seemed very soft, but very little tissue was removed by the curettage. Recovery was uneventful. The packing was removed gradually, and all was out on the fifth day. The bleeding had entirely ceased.

Pathological Findings.—The curettings contained many spindle and polyhedral-like cells, with numerous myotic figures. Very little normal endometrial tissue was found. A diagnosis of mixed-cell sarcoma was rendered by the pathologist.



(A.A.A.A). Tumor emboli.

This condition was carefully explained to the patient, and after some delay she requested me to perform a second operation; so on June 30th, under ether anesthesia I made a transverse abdominal incision, four inches long and two inches above the symphysis. This incision was carried through the anterior sheath of the recti, the sheath was freed from its attachments to the recti and linea alba above and below the incision for about two inches. The recti were then separated and a longitudinal incision made through the peritoneum. With the patient in a high Trendelenburg's position, this incision gave me a very good exposure of the pelvic contents. I now made a careful inspection of the uterus and adnexa, but discovered nothing abnormal save for a moderate enlargement of the uterus. On palpation I could feel no tumor masses nor any enlargement of the retro-peritoneal lymphatics.

However, in view of the pathological findings, I did a complete hysterectomy. I ligated and divided the utero-sacral, infundibulo-pelvic, broad, and round ligaments. This permitted me to draw the uterus well up through the abdominal incision. I now freed the uterus and upper third of the vagina from the bladder and ureters. This having been completed, it now only remained for me to clamp and divide the vagina, thus removing en masse the uterus, adnexa, and upper third of the vagina. I could discover no enlarged lymph nodes, so I closed the vaginal wound, leaving in a small gauze drain, and sutured the edges of the peritoneal wound over this drain. The abdominal incision was closed and the patient returned to the ward. She recovered from the anesthetic in good condition, and from then on made a very smooth convalescence with only a slight post-operative elevation of temperature and pulse, which promptly fell to normal on the third day after operation. On the tenth day after operation she was allowed out of bed, and on July 22d she was discharged cured.

PATHOLOGY OF SPECIMEN.—(For the following very interesting pathological report I am indebted to Dr. Larkin, Pathologist to the City Hospital):

Macroscopic Examination.—The uterus is enlarged, especially the body of the organ. It is of firm consistence, and on one section the following macroscopic points are seen: The endometrium is pale; partially edematous. It is firmly adherent to the underlying muscular structure, and cannot be moved readily over it with forceps. Walls of the caverna sinus are thickened, diminishing the lumen. Here and there are red, irregular, elevated areas with blood clots adherent. The muscular wall seems to be infiltrated with white irregular strands of denser consistence than the normal musculature. This condition is most marked throughout the body of the uterus; wall in the cervical portion shows no gross changes are to be seen. There is no inflammation or swelling of the round ligaments. Sections taken from different parts of the body of the uterus were hardened in different hardening fluids and sectioned for microscopic examination.

Microscopical examination of tissue reveals the following: There is al-

most complete absence of the glandular endometrium. Only here and there a few utricular glands can be seen. Normal stroma of the endometrium is absent, and in its place young vascularized and embryonal tissue-like cells are present. These extend throughout the whole endometrium body of the uterus. In this young proliferating tissue can be seen many myotic figures. The character of these cells for the most part are spindle and polyhedral-like cells; some with elongated nuclei, with dark staining chromatic bodies in the nuclei, and in the polyhedral-like cells chromatic material not quite so abundant. There is superficial necroses of the tumor tissue, in which it is quite impossible to make out the outline of the cells. Tumor tissue has infiltrated down into the deeper muscular structures, and is to be seen in an irregular network invading the medium-sized blood-vessels and capillaries. In several sections tumor thrombi can be seen in the larger blood sinuses. From these tumor emboli radiate out irregular, black lines, which are evidently blood-vessels filled with compressed tumor cells. One noteworthy feature microscopically is the character of the larger blood sinuses, which show marked hyaline degeneration of their walls, staining bright red with eosin. The lumen of these sinuses is almost entirely obliterated. Several sections of these abound in knobs, or clusters.

The type of cells making up the tumor is that of the mixed-cell sarcoma, showing very early phase. Various stains used show the cells arranged in irregular areas separated one from the other by a very delicate stroma. This tumor of the uterus is interesting for the following points:

First, the complete absence of the endometrium and its replacement by a vascularized embryonal connective tissue of mixed type. Second, the almost complete absence of uterine mucous membrane. Third, the infiltration of the musculature by strands of tumor tissue invading the blood spaces, localizing themselves in several of the larger blood spaces as tumor emboli, giving a more or less complete network, as shown in the photograph. Fourth, the hyaline degeneration of extreme degree of the walls of the caverna sinuses, almost occluding their lumen. Microscopic examination of the outer musculature shows no apparent change, so that the lesion is one which involves the inner third of the uterine cavity.

Diagnosis.—Early sarcoma of uterus, mixed-cell type tumor emboli of blood-vessels, and larger sinuses.

I last saw the patient on July 15, 1914, and at that time she was in good general condition, there being no evidence of any recurrence. She had suffered at intervals from some of the symptoms of induced menopause, but was otherwise perfectly well.

CONTRIBUTIONS TO THE TREATMENT OF LONG-
BONE FRACTURES

BY

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CONTRIBUTIONS TO THE TREATMENT OF LONG-BONE FRACTURES

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I. STEINMANN'S NAIL EXTENSION IN FRACTURES OF THE FEMUR¹

The essential part of Steinmann's² method consists of a *nail* which transfixes the soft parts and bone, and which is the *sole point of attachment for suitable traction upon the lower fragment*. In other words, the bone and soft parts are transfixed by a drill which is left in place with both its ends projecting.

Apparatus. The steel nail is $3\frac{1}{2}$ mm. thick, 18 to 20 cm. (7 to 8 inches) long; the ends should project $2\frac{1}{2}$ to 3 cm. beyond the skin (Fig. 1). The nail may be driven through the bone by a bit and brace, or by a geared hand drill (Fig. 2). An adjustable pair of tongs (Fig. 3), the limbs of which are joined by a suitable thumbscrew, forms the most convenient means of attaching extension apparatus to the nail ends.

In describing the typical procedure, let us take, for example, the treatment of a fracture of the femoral shaft at its middle third. The sterilized nail and drilling mechanism are fitted together and placed in readiness. The entire limb is brought close to the margin of the table or bed, so as to prevent interference with the drilling that is to follow. One assistant grasps the patient's ankle and, maintaining traction, elevates the leg, while the operator who is "sterile," applies full-strength tincture of iodine to the skin and does the necessary draping. The leg is then lowered, so that ankle and knee are close to the edge of the table. Another assistant having sterilized himself, assumes management of the broken limb. The anesthesia (gas) is now started. The assistant steadies the limb (outer margin of the foot must stay vertical), at the same time pulling the skin of the thigh upward. The point of the nail is inserted horizontally and at right angles to the shaft *a finger's-breadth above the upper margin of the external condyle*. As soon as the bone is touched, drilling is begun (Fig. 4). The operator controls the drill with both hands and leans his body, either chest or groin, against the breast-piece of the drill. The resistance which at first

¹ Taken in part from my article in the American Journal of the Medical Sciences, August, 1913, No. 2, vol. cxlvi, p. 157.

² Nagclexension der Knochen brüche, Neue Deutsche Chirurgie, 1912, Band i; British Med. Jour., November 30, 1912, p. 1235.

is encountered lessens after a while, for penetration is easier after the corticalis is passed, as the cancellous tissue offers less resistance. Again, progress is slower as the corticalis of the opposite side is encountered. Finally, this also is passed and the nail point may be felt emerging from

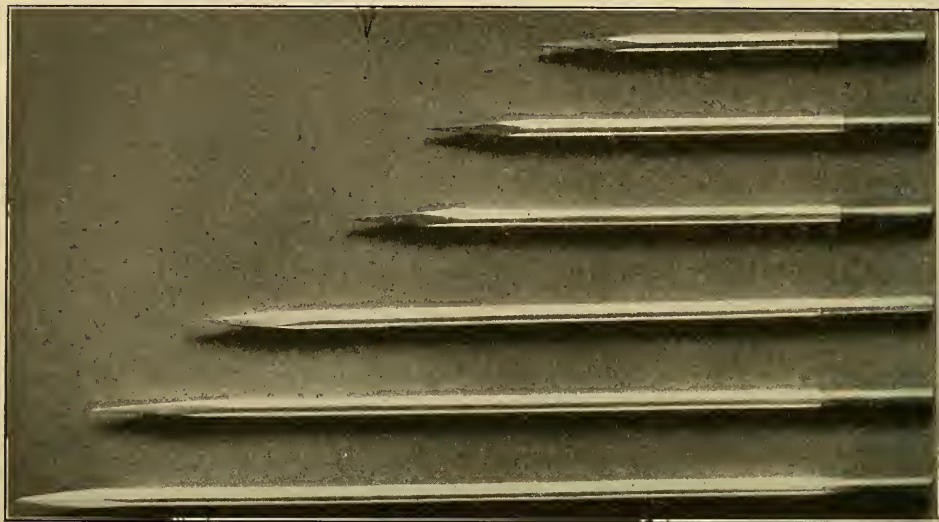


FIG. 1.—Solid nails of varying lengths. The points are sharpened on four sides; the heads are round in order to fit the chuck of the hand drill.

the bone. The skin overlying the nail point is pulled upward by the assistant and the point finally emerges. Drilling is stopped when the two nail ends project equally on either side of the limb. The narcosis is then

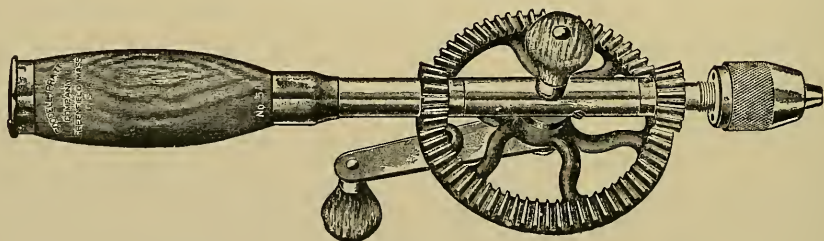


FIG. 2.—Geared hand drill.

stopped. The skin which the penetrating nail point has carried away from the leg is now pushed back on the nail to the level of the adjacent skin. Small flat pieces of folded gauze are transfixed by the nail points and run down the nail shaft to the skin.³ Drawing these aside, aristol and lastly collodion are applied to the skin where the nail emerges. A figure-of-eight

³ Another nail may be used to make holes in the gauze, so that it readily slips over the rounded head of the nail which is in place.

bandage holds them in place; it is well for some turns of the bandage to be transfixed by the nail points.

The sides of the nail's point are clogged with accumulations of caked bone and marrow particles. These should be scaled off as soon as the dressing has been completed. If this is neglected, these accumulations will dry in place and will then be hard to dislodge—the subsequent sterilization of the nail point just prior to withdrawal will be tedious, difficult, and uncertain.

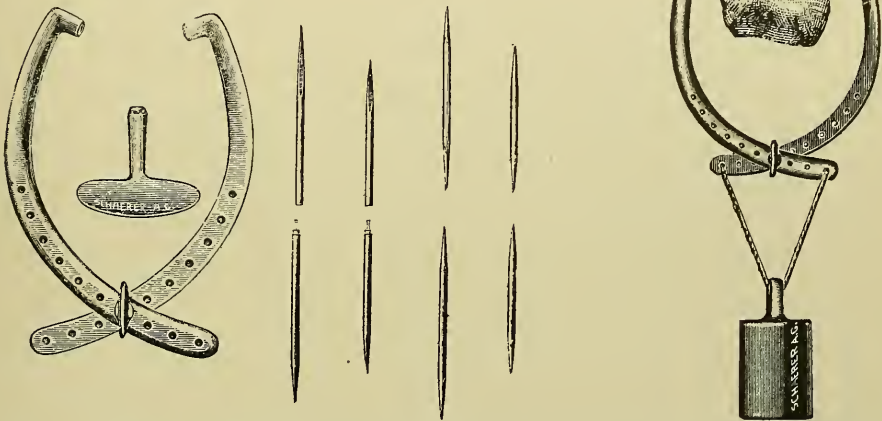


FIG. 3.—Tongs; two piece nails; one piece nails (very short); also schematic attachment of nail extension in the supracondylar position.

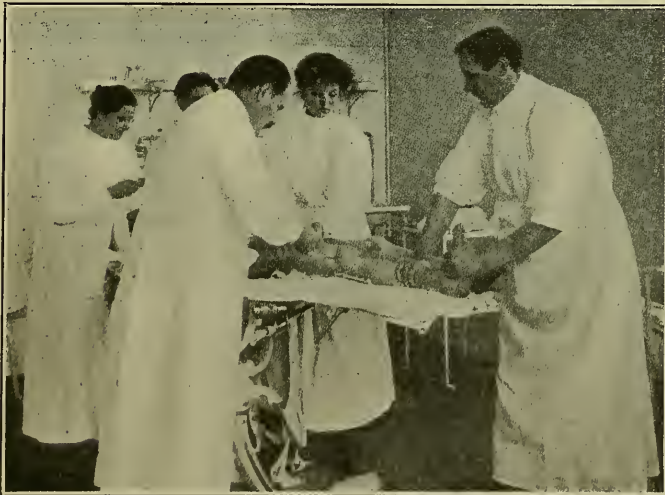


FIG. 4.—Transfixing the lower end of the femur. (Steinmann.)

The extension apparatus is now attached to the nail. The two limbs of the tongs are applied so that the thumbscrew holding them comes uppermost—each limb is applied separately—the screw being inserted in that hole

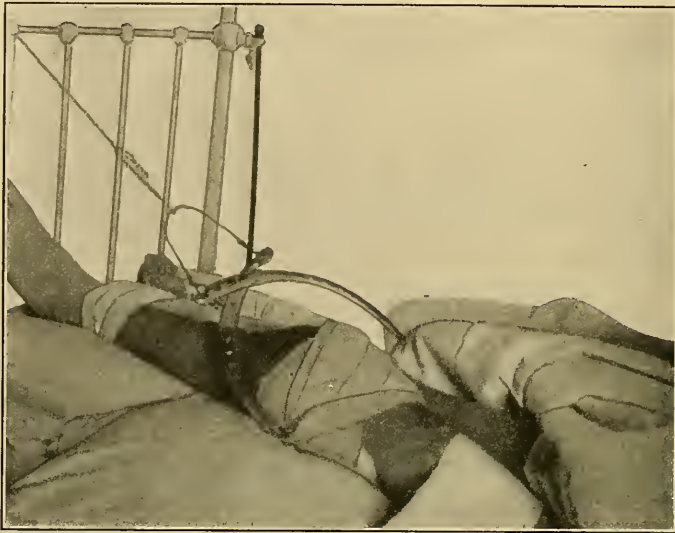


FIG. 5.—Supracondylar attachment of nail extension. Note wire fastened to ends of crossed limbs of tongs.



FIG. 6.—Note traction wire in line with axis of thigh; also suspension of outer end of nail to prevent eversion (rotary displacement).

of either limb which most readily falls in alignment. The ends of a short piece of wire are fastened through the outermost holes of the crossed limbs of the tongs (Fig. 5). At the center of this wire is attached the main traction wire or rope leading over a pulley at the foot of the bed down to

the suspended weights. The thigh is placed in semiflexion. The leg is horizontal or the foot may be lower than the knee. The heel should not touch the bed. Hard pillows, sandbags, or suitable splints may be employed to maintain this position. The pulley at the foot of the bed must be placed so that the wire leading from the tongs is in line with the axis of the semiflexed thigh (Fig. 6).

Naturally the amount of weight to be used depends upon the individual case. In a recent fracture of an adult femur, with 5 cm. shortening, 18 to 20 pounds are usually adequate. It is very convenient to use a small spring scales registering up to 40 pounds, to ascertain how much weight is being applied (Fig. 7).

Countertraction may be obtained by (a) raising the foot of the bed; (b) a well-padded perineal loop attached to the head of the bed, and (c) a foot-rest for the sound limb.

Shortening is usually overcome within a week. The broken limb should be allowed to stretch 1 cm. longer than its fellow. This renders proper approximation of the fragments easier. In transverse fractures, after proper reduction, overcorrection is no longer necessary, while in oblique fractures it is well to maintain $\frac{1}{2}$ or 1 cm. overcorrection until union takes place.⁴ After the shortening has been overcome, less weight is required to maintain the fragments in proper position. Undue lengthening is readily corrected by simply lessening the weight—the limb promptly shortens. *Massage and passive motion may be begun five days after nail-extension has been instituted.*

At the end of three or four weeks there usually is sufficient callus to justify removal of the nail. Steinmann states that, if possible, nail-extension should not be maintained longer than three weeks, and never longer than five weeks. Thirty-four days is the longest time I have personally seen the nail-extension maintained.

Extension is discontinued, dressings are removed from the inner (point) end of the nail hole. The projecting point of the nail and the adjacent skin are thoroughly cleansed with iodine. The laughing-gas anesthesia is now begun—it is discontinued the moment the nail is out. Grasping the rounded head with a heavy pair of wire pliers, the nail is first *rotated* to loosen it, and is then extracted with a rotary motion. The knee should be steadied by an assistant. Under gas, the actual extraction takes less than one minute. Some tincture of iodine is injected into the nail holes and a wet dressing is applied, until the skin is healed—usually within five or six days.



FIG.
7.—Scales.

⁴ According to the painstaking investigations of Wettstein (Beitr. z. klin. Chir., 1908, Band ix, Heft 3, p. 684, and Korrespondenzbl. f. Schweizer Aërzte, 1909, No. 3, p. 73) fractured limbs shortened $\frac{1}{2}$ to 1 cm. within the year after union had occurred. To counteract this we institute overcorrection in oblique fractures.

The subsequent treatment is that appropriate for any limb with a fresh callus—namely, avoidance of too much strain on the callus, at the same time employment of suitable passive and active motion.

I. DETAILS OF TREATMENT

A. *Location of the Nail.* According to Steinmann (1) it should avoid the hematoma at the site of fracture; (2) it should avoid the marrow cavity⁵; (3) it should avoid the joint capsule; and (4) it should avoid the epiphyseal line. Hence the nail is inserted one finger's-breadth above the upper margin of the femoral condyle just behind the middle line of the bone—in other words, through the posterior half of the supracondylar region just anterior to the adductor tubercle or its corresponding site externally.

In fractures of the lower third of the femur the nail is inserted through the upper end of the tibia $2\frac{1}{2}$ fingers'-breadth below the upper margin of the tibia on a level with the lower margin of the head of the fibula (Fig. 8).

B. *Methods of Inserting the Nail.* (1) Hammering has been found to cause too much pain at the site of fracture—the risk of splintering the bone is not great.⁶ (2) Boring by hand is safer than by electricity. (3) Incision of the soft parts is unnecessary and increases the likelihood of infection. Other modifications of the method, such as boring a hole and then inserting the nail, etc., can be placed in the same category. (4) It is necessary to pull the skin upwards, especially in supracondylar insertion of the nail, because if this is not done, as the limb is stretched, decubitus of the skin will occur to the distal side of the nail.

C. *Type of Nail.* Both head and point of the original nails are squared. They do not accurately fit the ordinary hand drill with a three-point contact chuck, hence I use nails with round heads which fit the usual types of drill (Fig. 2).

The two-piece nail apparently cannot be made of sufficient strength to afford a proper factor of safety against breakage at its joint. The nail has broken six out of the sixteen times I have used it. The weights used were never more than 20 pounds, usually about 15 pounds. Fortunately, when the tongs were used the broken nails were held in sufficiently good position to still maintain traction (Fig. 25). Breaking has occurred during insertion, during transport of the patient in bed to the x-ray room, and with the patient lying quietly in bed as late as the eighteenth day. I therefore prefer solid nails.

D. *Dressing.* Dressing should be dry and rather voluminous. The details of application have already been given.

E. *Attachment of Traction.* The tongs are most convenient for this purpose, but they are not essential. Braided picture wire, copper wire, or

⁵ At the point where the nail passes through the bone, there is enough cancellous tissue to prevent direct communication with the main marrow cavity.

⁶ In a recent case of Dr. William H. Bishop's it was impossible to insert the nail by hand drilling because of the extreme hardness of the femur. The patient was a woman, aged fifty years, with a fracture of the upper third of the bone. A hammer was not available at the time, so the attempt to insert the nail had to be given up. At the subsequent plating the bone was found to be just as hard at the site of fracture, as it was near the condyles. The corticalis was very thick and there was cancellous tissue in the marrow cavity.

bandages (Figs. 9 and 14), may be used. In this case the ends of the nail are protected by corks. Spreaders may be necessary to prevent undue pressure of the wire against the skin in some instances, while in others an S-hook or wire loop may be required to prevent the end of the traction wire from slipping off a nail end. Where tongs are used, the traction should be made upon the ends of the crossed limbs, not at the point where they cross (Fig. 6).

F. *Anesthesia.* There may be a short general anesthesia, either laughing-gas or "æther rausch," or there may be a local anesthesia (0.5% novocaine) in the cases where the former is contraindicated. In intelligent adults it is possible to do without an anesthetic, for the pain of the boring is by no means unbearable. Indeed, most of the pain is felt at the site of fracture.

G. *Amount of Weight.* In recent fractures this varies between 10 and 30 pounds; in old fractures between 20 and 60 pounds. Steinmann speaks of one case where an old fracture with malunion was refractured, nail-extension was applied, and a lengthening of 11 cm. was obtained within eight days. As said before, most of the stretching is done in the first week, after which less weight suffices to maintain proper position.

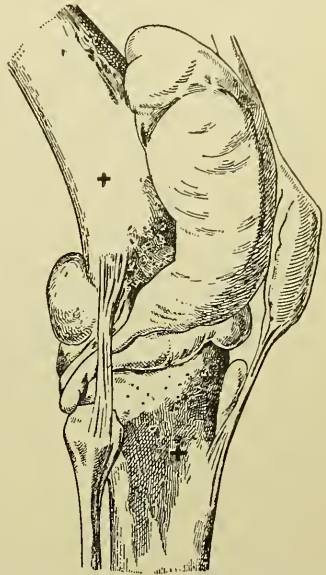


FIG. 8.—Crosses indicate proper locations for insertion of nails in the neighborhood of the knee joint. (Steinmann.)

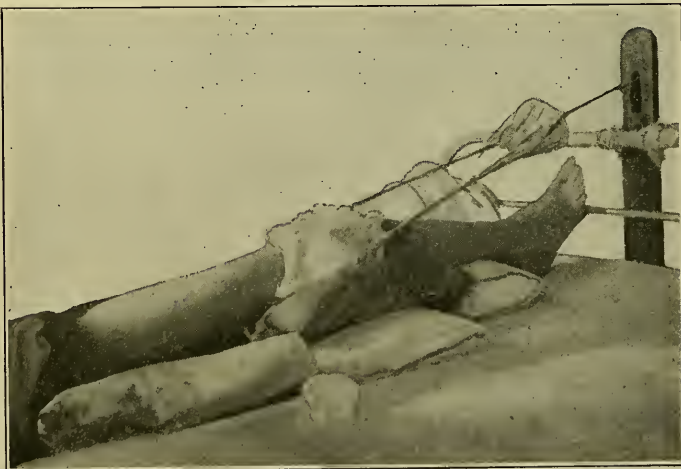


FIG. 9.—Pertrochanteric fracture (Case II). Tongs were not available, so extension was made by attaching bandage material made into rope to the projecting ends of the nail. Note limb in semiflexion with small sandbag under outer end of nail to prevent eversion.

H. *Control of Fragments.* The direction of the pull can be varied to adapt the lower to the upper fragment. Strong traction often causes the upper fragment to follow the lower to a great extent—that is, lateral dislocation is corrected largely by extension alone.

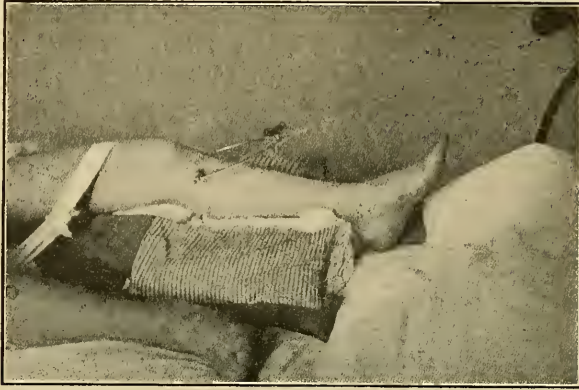


FIG. 10.—Nail extension applied to head of tibia. Used in cases of supracondylar femoral fracture. (Steinmann.)

Posterior dislocation of the lower fragment in supracondylar fractures is known to be especially difficult of correction. Flexion of the leg upon the thigh and dorsal flexion of the foot upon the leg lessen the pull of the

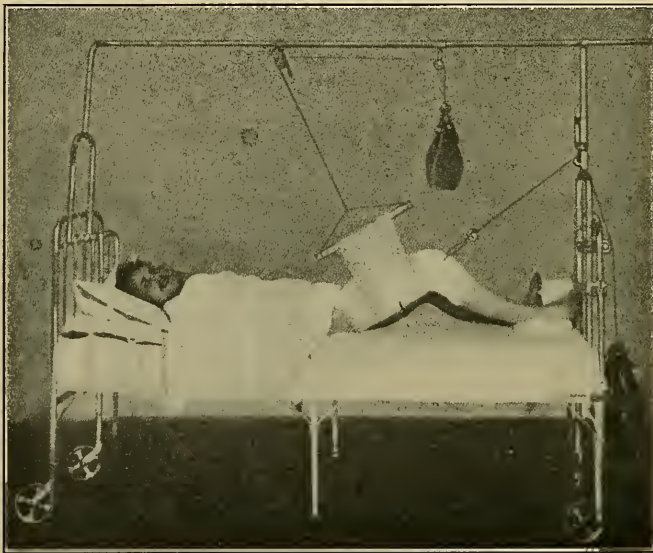


FIG. 11.—Nail extension with anterior pull at right angles to the limb. (Wægner.)

gastrocnemii upon the lower femoral fragment which is displaced backward. Traction at right angles to the leg also aids in correction of this condition (Figs. 10 and 11).

Rotary displacement (eversion) is best corrected by suspension of one nail end. This is most conveniently accomplished by two uprights and a horizontal bar running the length of the bed, with a wire attached to the lateral (outer) end of the nail and to the horizontal bar (Fig. 7). In fact, the entire limb may be most conveniently suspended by this simple means.

It is unnecessary to review all the modifications and varieties of lateral traction and suspension which have been employed in other extension methods, notably in that of Bardenheuer. It goes without saying that they have been employed in the Steinmann method since 1907 with even better chances of success.

X-ray. If possible, X-ray plates should be made before extension is applied; afterward controls can be made at proper intervals to make sure of proper position. As a matter of course, the exposures should be made both from in front and from the side.

The ideal method is to bring the X-ray apparatus to the bedside and to

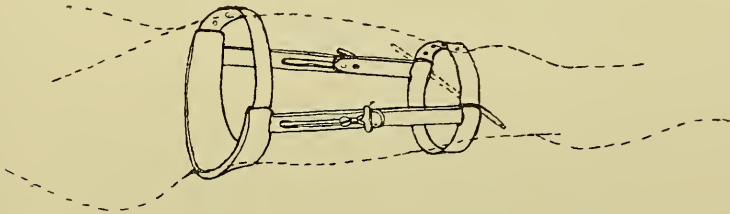


FIG. 12.—Steinmann's splint for maintaining extension during transport.

take exposures without disturbing the fractured limb. Very few hospitals have this convenience. Another way is to move the patient, bed and all, to the X-ray room by means of a bed truck. In many institutions this also is not feasible; either there is no bed truck, or the elevator is too small to accommodate the bed, or the doors are too narrow. Besides these objections, such transportation is most disagreeable to the patient unless it is made very slowly, because the slightest jar is promptly transmitted to the fractured limb in magnified form by the weights and pulleys of the traction apparatus.

Lastly, a suitable splint may be employed, by means of which extension is maintained during transportation on a stretcher from the ward to the X-ray room. The splint used by Steinmann for this purpose (Fig. 12) has certain faults which I have endeavored to eliminate. I use a long hip splint (Fig. 13a and 13b), reaching from the pelvis well beyond the heel. The upper padded ring is made in two halves which are hinged behind, while their free ends in front are locked with a thumbscrew. The lateral longitudinal bars are made of hard wood so as not to interfere with obtaining lateral exposures by the X-rays. The metal stirrup or crossbar joining the lower extremities of the longitudinal wooden bars is composed of two over-

lapping slotted parts held together by a thumbscrew. A semicircular support for the tongs is described below.

The splint is applied in the following manner: The thumbscrews at either end of the splint are loosened so that the oblique pelvic ring (heavily padded) is opened and its jointed halves are accurately fitted in place, the patient merely raising himself a little. As the ring is closed, the longitudinal bars and stirrup are adjusted. Slings of bandage material are passed behind the limb, at the middle of the thigh, the knee, and the ankle. A metal arch with ends resting on the longitudinal bars supports the Steinmann tongs and prevents them from impinging against the shin. Finally, the rope connecting the tongs and weights is firmly grasped and the weights are removed. Without lessening the traction, the rope is fastened to the cross-

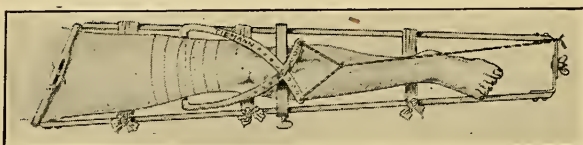


FIG. 13a.

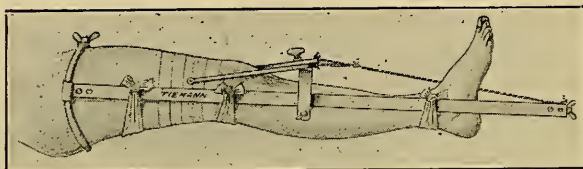


FIG. 13b.

FIG. 13.—The author's splint for maintaining extension during transport.* The longitudinal side bars are of wood instead of metal to facilitate lateral X-Ray examination in the taking of lateral exposures with the X-Ray.

Fig. 13a, front view; Fig. 13b, side view.

bar or stirrup. Once this is done, the entire limb may be moved by lifting the splint and this without causing pain or displacing the fragments.

Even when the X-rays are not available, very fair results can be obtained by careful daily control with tape measure and fingers. While measurement is being made, the sound limb must be flexed to the same degree as the fractured one. It is well to measure from a number of points for the sake of control—for example, from the anterior spine to the nail, as well as to the internal malleolus.

II. OBJECTIONS WHICH HAVE BEEN MADE REGARDING STEINMANN'S METHOD

A. *Pain.* (1) Upon introduction of the nail, an anesthetic is usually employed, yet under unusual circumstances it may be dispensed with.

* From the American Journal of Surgery, January, 1914.

(2) During extension, no pain is felt at the site of the nail. If traction is irksome, the weights may be lessened for a few hours and then added again.

(3) At removal of the nail, there may be slight pain. It is well not to allow children or excitable adults to know that a nail is being employed. As long as they are ignorant of its presence, they do not complain. A voluminous dressing applied to the leg satisfactorily conceals things.

B. *Infection* occurs either at the time of insertion or after two or three weeks. It is easy to insert the nail aseptically, but it is difficult to keep the region aseptic in the later stages. The granulations which have formed by the end of three weeks afford a fairly good protection to the bone at large. Small local infections have been known to occur, especially in long-continued cases of extension. These have all remained localized. Small circular sequestræ have come away. A small exostosis occasionally develops at the site of the nail. So far, *there has been no mortality from nail-extension carried out according to the rules laid down by Steinmann.* Fatal cases *are* known where the wound made by the nail has established communication with the fracture hematoma or with the knee joint. In a series of over 33 cases, I have only seen one instance where a bone fistula followed the application of nail-extension. Very small pads of gauze with a very narrow bandage were used; the bandage slipped behind the gauze in the course of a few days—that is, it lay between the gauze and the skin in direct contact with the nail and the wound. A suppuration which remained strictly localized occurred; the acute stage promptly subsided under appropriate measures, but the fistula has persisted for more than nine months.

In about a third of the cases, the wound is absolutely dry at the time of the nail's withdrawal—it looks as if the insertion had taken place the day before instead of three or four weeks; in the remainder, there are a few drops of purulent secretion around the nail, but no reddening of the adjacent skin. The fact that such wounds heal within a week and remain healed, is proof that the few drops of thin pus did not contain virulent pathogenic organisms. Wounds of this sort must be treated with the same scrupulous asepsis accorded to those which are perfectly dry.

Most of my cases ran a slightly elevated temperature, between 99 and 100 degrees, while the nail was in place; this dropped again upon removal of the nail.

C. *Injury by the nail to important nervous, vascular or articular structures.* There has been none at the time of insertion. Steinmann even reports a case where the nail had been run through the ankle-joint without causing any functional trouble.

The epiphysis has not been harmed, as shown by four years' observations upon children on whom this method had been employed. There was no lessened growth of the injured side. (Steinmann.)

D. *Injury by too great traction* has been ascribed to nail extension. There has been no instance of harm to the soft parts. An extreme example

of what can be accomplished is shown by the case of Steinmann's, where 11 cm. lengthening was obtained within seven days in a refractured malunion of five or six months' standing. There was no loss of function.

E. *Lax Knee-joint.* Strong diversity of opinion exists regarding injury to the knee-joint by traction (such as Buck's extension) applied to the limb below the knee. Naturally this controversy will promptly transfer itself to application of the Steinmann method in fractures of the lower third of the thigh where the nail is inserted through the head of the tibia; hence, a brief outline of the situation seems proper:

Certain observers emphatically state that they have seen the most harmful effect upon the knee-joint from extension applied to the leg below the knee. Other equally competent authorities maintain that they have never seen any injury to the knee-joint from application of Buck's extension to the leg as well as the thigh, and furthermore insist that no adequate extension can be obtained by merely pulling on the skin of the thigh.

It is to be noted that Steinmann's method differs from Buck's extension in that the knee-joint is flexed, whereas in Buck's extension the knee-joint is fully extended. Perhaps there is less strain upon the ligaments when traction is applied to the partially flexed joint than when there is full extension. My own experience in this matter is not sufficiently extensive to be of much value. Steinmann states that he has never seen any distraction of the knee-joint from traction upon the tibia. He also states that effusions into the joint disappear with great rapidity after extension is made; this likewise has been my own experience. The transient inability fully to extend the knee rapidly disappears under appropriate massage and passive motion.

F. *Delayed Union.* None has been observed so far. On the contrary, Steinmann says that firm union is usually obtained in seven or eight weeks; in some cases even as early as five weeks. The fact that both passive motion and massage can be employed while extension is going on certainly has much to do with prompt callus formation and union. No pseudarthroses have occurred.

III. ADVANTAGES

A. *Traction.* Less weight is needed for any given case than by other methods. More weight can be used than by any other method, and this without the risk of decubitus. The traction is continuous, not spasmodic, as in Codivilla's method or in Lane plating. In cases of long-standing malunion, the soft parts may be so contracted that, after freeing their ends, the bones cannot be placed end-to-end without undue tension upon the soft parts, which may cause cessation of circulation in the distal part of the extremity or even rupture of important structures. It is in such cases that the continuous traction safely accomplishes its purpose in from three to five days.

B. *Painlessness.* Once the nail is in position, there is no pain due to its presence. In fact, after a few days the patients are apt to become careless because of their freedom from pain.

C. *Control of the fragments,* especially of the lower one, is more certain by this method than by any other except the open operation.

D. *The small site of attachment* makes the method available in the compound fractures where other traction methods are either difficult or impossible of application.

E. *Availability at comparatively late periods.* The fact that nail-extension can be employed with success at a considerable time after the fracture has occurred makes it of value when a fracture is complicated by extensive abrasions of the skin or by acute disease, such as delirium tremens or pneumonia. Here one can wait until recovery has taken place before applying extension. The method has been successfully used in fractures of forty days' standing in which usual traction methods have been of no avail.

F. *Simplicity and possibility of improvisation* are qualities of the method which cannot be too strongly praised. Steel drills and the apparatus for driving them, or long nails, together with some wire or stout cord, are obtainable almost anywhere, and they can be made to serve one's purpose.

The value of the method in military or naval service or in inaccessible regions is readily apparent. The Prussian army uses it. It is time-saving, effective, and easily learned.

Comparison of Methods. With Buck's extension we have more or less shortening and stiffness of the knee, which lasts for a long time afterward. With the Steinmann method there is no shortening, and the transient inability fully to extend the knee-joint quickly disappears. In some cases it is never present.

With Lane plating we have the risk of grave infection if the operation be done by any except the skilled, and in any case, somewhat delayed union. In certain exceptional cases even with no infection, union may be delayed for months, and long immobilization of the knee-joint in such cases has resulted in what amounts to an ankylosis. With the Steinmann method there is much less risk of infection (there have been no fatal cases so far); there is no delayed union (in the average case), and the joints have little, if any, impaired function.

The method of Ransohoff,⁷ whereby the point of a pair of ice-tongs are lightly driven into the bone, is a recent modification of the same principles governing nail extension. The site of attachment with tongs is larger than with the nail.

⁷ Lancet-Clinic, August 17, 1912.

IV. INDICATIONS FOR USING NAIL-EXTENSION

Indications for using nail-extension can be summed up in the phrase, "When the usual traction methods fail."

A. *Recent Fractures.* Fractures of the upper end of the femur constitute debatable ground.

I have had no experience with intracapsular fractures, and am therefore not in a position to judge of the relative merits of the various existing methods. On the other hand, I have been able to obtain excellent reduction by employing nail-extension in pertrochanteric fractures.

In fractures of the upper one-fourth of the shaft, a great deal of care and ingenuity must be exercised to bring the lower fragment into proper alignment with the upper, because of the latter's well-known tendency to remain pointing forward and outward irrespective of the amount of traction employed—in other words, in this situation traction *per se* does not overcome lateral displacement. The lower fragment must be brought in line with the upper by bringing the thigh into flexion, abduction, and a little outward rotation, in which position traction is now applied. Lateral traction may also be needed.

In fractures of the shaft, in very obese individuals where adhesive plaster is not effective, nail-extension is indicated.

In supracondylar fractures it affords better control of the lower fragment than any other traction method.

A case of von Eiselsberg's, quoted by Steinmann, illustrates this. In a fresh fracture of the lower third of the femur it was found that there was such pressure upon the popliteal artery that the pulse was obliterated. Traction by the usual adhesive plaster did no good. Nail extension was then resorted to and the pulse returned.

B. *Old Fractures.* This is a field which has been practically untouched by traction methods.

In malunion with much overriding, operation followed by the Steinmann method is the only way in which an ideal result can safely be obtained. The malunion is broken up, and afterward nail-extension is applied. Naturally, larger weights (up to 60 pounds) must be employed. This procedure has been used as late as three to six months after malunion had taken place.

In pseudarthroses, rubbing of the bone ends together, followed by application of extension, has been suggested. When this is not sufficient, the bone ends may be cut down upon and freshened, the wound then closed and nail-extension applied.

As a preliminary to Lane plating in cases where that method is indicated (interposition of soft parts, etc.), nail-extension is excellent, because the absence of shortening lightens the operator's task fully 75 per cent.

To conclude, in certain cases nail-extension is the only method to be used; in others, it is an aid to previously existing methods.

I take the opportunity of expressing my gratitude to Drs. Howard Col-

lins, William H. Bishop, A. G. Gerster, John A. Wyeth, Seth Milliken, and Ernest Fahnestock for their kindness in allowing me to employ this method in their services and for the privilege of publishing their cases. The abstracts of those treated in the City Hospital are appended.

SYNOPSIS OF CASES

CASE I. James L., aged four and one-half years (Dr. Howard Collins' service, City Hospital, Blackwell's Island).

June 26, 1912. A board fell from a building and struck the patient, causing an oblique fracture of the middle third of the left femur. Admitted to City Hospital on June 27.

July 2. Sixth day after fracture. Steinmann two-piece nail; supracondylar insertion. Laughing-gas anesthesia; 6 pounds' traction; 1 cm. shortening.

July 9. $\frac{1}{2}$ cm. overcorrection; 4 pounds' traction.

July 22. $\frac{3}{4}$ cm. overcorrection.

July 23. Twenty-first day. Nail removed. (X-ray, Fig. 14.)

July 25. Limbs equal; nail holes clean.

August 3. Child can raise entire limb to vertical position; flex and extend knee.

Nail holes, two small, clean, granulating spots.

October 5. Function perfect; runs and walks without a limp.

November, 1913. No shortening, no bowing, perfect function.

Epicrisis. This was a restless, active child, who had no pain after the



FIG. 14.—Case I. Twenty-one days after fracture; no shortening; spiral fracture.

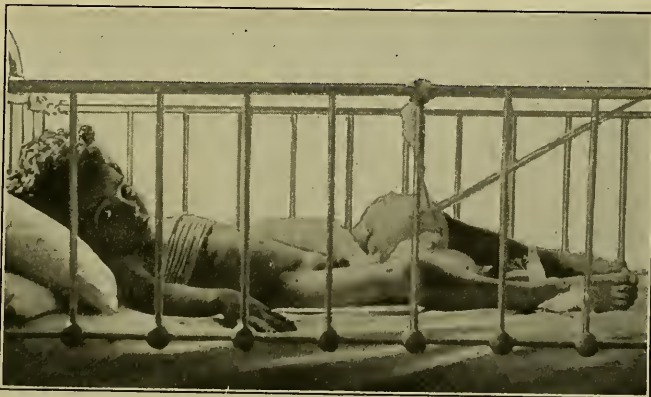


FIG. 15.—Case I. Illustrating restraint necessary with active children to maintain proper alignment of fragments.

first three days, and who had to be restrained by loops of bandage at the ankle, knee, hip, and armpit in order to maintain proper alignment of fragments (Fig. 15).

CASE II. James R., aged sixty-five years (Dr. Howard Collins' service, City Hospital, Blackwell's Island).

June 14, 1912. Admitted to Bellevue Hospital with a fracture of the neck of the femur.

June 21. Transferred to the City Hospital with a shortening of $1\frac{1}{2}$ inches and marked eversion.

June 22. Buck's extension with 10 pounds' traction applied.

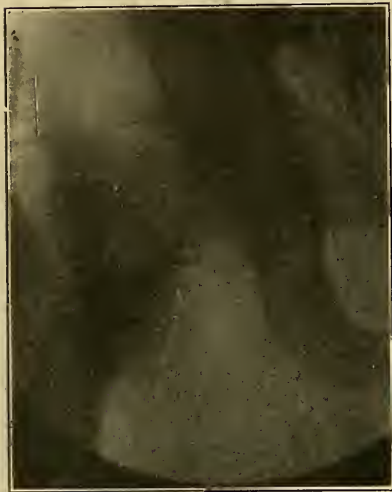


FIG. 16.—Case II. Pertrochanteric fracture 19 days after application of nail extension.



FIG. 17.—Case II. The same 5 months later.

July 1. Sixteenth day after fracture, 3 cm. shortening. Steinmann two-piece nail; supracondylar insertion under laughing-gas; 21 pounds' traction.

July 12. $\frac{1}{2}$ cm. shortening.

July 18. Eighteenth day after insertion. Nail broke at middle; inner half loose and removed; wet dressing applied to nail hole. Outer half was solid, and sandbag, which had been placed underneath its end to prevent eversion, and to maintain the limb in proper position, was left undisturbed.

July 19. 1 cm. shortening; X-ray plate (Fig. 16).

July 23. Outer half of nail removed.

July 27. Nail holes healed; good union; massive callus.

July 29. 2 cm. shortening; patient comfortable.

August 14. Patient is encouraged to sit up and to try to walk on crutches.

August 28. Shortening nearly 3 cm.; good flexion and extension; some limitation to inward rotation. Edema of right leg and ankle, which has existed since patient was out of bed on crutches, gradually subsiding under massage.

December 1. Patient has been on crutches for the past three months. Has now been encouraged to walk.

December 23. Bad dermatitis of right leg and ankle from unwise use of chloroform liniment.

December 30. X-rays (Fig. 17) show neck joined to the shaft at more of a right angle than previous picture; firm union.

Epicrisis. The nail broke on the eighteenth day. Traction was being made by bandages fastened to the projecting ends of the nail. No tongs were available at that time, and it was not feasible to apply other methods of traction. Had extension been maintained for a week longer, it is probable that no shortening would have ensued after removal of traction. Where the two-piece nail has broken with the tongs grasping its ends, it has been found possible to still maintain traction, since the tongs hold the fragments of the nail at approximately right angles (see Case VI).

CASE III. Mary H., aged seven years (Dr. Howard Collins' service, City Hospital, Blackwell's Island).

August 24, 1912. Fell three stories to the ground from a fire-escape, striking a shutter and other fire-escapes on her way down, sustaining a fracture of the lower third of the right femur. A projecting fragment could be felt in the popliteal space. There was no obstruction to the circulation; 3 cm. shortening.

August 26. Second day after fracture. Under laughing-gas anesthesia. A two-piece nail driven through the head of the tibia; 8 pounds' traction.

August 28. $\frac{1}{2}$ cm. shortening.

September 3. X-rays show lower fragment displaced outward (Fig. 18).

September 9. Reduction of fragment under laughing-gas, by Dr. A. L. Sherman.

September 19. Twenty-fourth day; nail removed.

September 26. Excellent union; patient can raise heel from bed, flex the leg completely, and extend it to within a few degrees of full extension.

October 10. X-rays (Figs. 19 and 20) show excellent position.

October 18. Out of bed for the past few days; can now fully extend leg; limps less and less as time goes on.



FIG. 18.—Case III. Supracondylar fracture before reduction.

October 19. Discharged from hospital.

November 14. Some effusion of knee-joint, which the mother says has been present since leaving the hospital. Limp barely perceptible.

December 24. Effusion of knee gone; walks and runs with perfect freedom.

Epicrisis. There was union within one month. Slight limitation to full extension, which was present for a time after removal of the nail, gradually disappeared. The effusion of the knee-joint was present for five or six weeks after discharge from the hospital, and subsided without treatment.

CASE IV. John H., aged eighteen years (Dr. Howard Collins' service, City Hospital, Blackwell's Island).



FIG. 19.—Case III. Both knees after reduction.

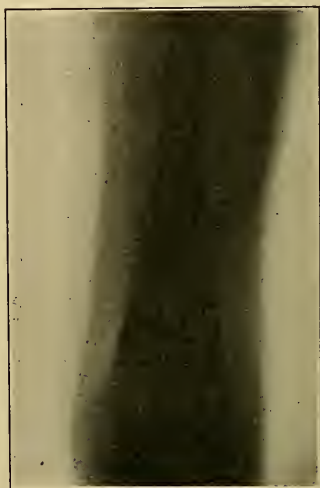


FIG. 20.—Case III. Final result.

September 30, 1912. Was hit by an automobile mud-guard while roller-skating in the street. He was knocked senseless, and sustained a fracture of the middle third of the left thigh.

October 1. A large effusion of the knee-joint. Shortening about 4 cm.

October 3. Fourth day after fracture, Steinmann's two-piece nail inserted through the head of the tibia; 10 pounds' traction.

October 5. Shortening 2.5 cm.; 18 pounds' traction. Effusion of the knee-joint gone.

October 10. 1 cm. overcorrection.

October 23. Twenty-first day. Nail removed. Some force necessary, because of its firm fixation in the tibia.

October 25. Plaster spica applied, reaching to the hip. At this time there was slight lateral mobility, considerable callus, and it seemed as though the upper fragment had moved anteriorly. There was slight lateral mobility of the knee-joint; no limitation to passive motion of the knee-joint.

October 31. X-rays (anteroposterior view) show good position (Fig. 21). Lateral view (Fig. 22) shows overriding of transverse fragments. This was the first time this case had been X-rayed.

November 6. Cast removed. Up in rolling-chair.

November 15. Patient slipped getting into chair and fell to the floor.

November 17. There was marked bowing and $2\frac{1}{2}$ to 3 cm. shortening. Supracondylar insertion of nail was suggested as the best available traction. This was refused by patient. Because of this, Buck's extension (15 pounds) and lateral traction (15 pounds) at the greatest prominence of the bowing were applied.



FIG. 21.—Case IV. Transverse fracture seen from in front.



FIG. 22.—Case IV. The same seen from the side. The plate taken at the same time as Fig. 21 was lost, but it showed the same position as this plate (Fig. 21), which together with Fig. 23 was taken 2 months later.

November 26. Bowing of thigh has gone; lateral traction discontinued.

December 1. Buck's extension discontinued; 1 cm. shortening present at this time.

December 22. Patient has lain quietly in bed the past three weeks, the leg being in the same position that it was while Buck's extension was being maintained. There was 2 cm. shortening. Flexion beyond 60 degrees limited. Full extension. Some effusion of knee-joint ever since discontinuance of Buck's extension.

December 24. X-rays show good callus and slight overriding of the fragments (Figs. 22 and 23). There is no angulation of the thigh.

December 30. Home on crutches, with a perfectly solid union.

Epicrisis. There were no facilities for controlling treatment by the

X-rays. Because of the extensive effusion of the knee-joint and because of the contusions of the thigh, it was deemed wiser to apply traction to the head of the tibia rather than to run the risk of possibly infecting a hematoma of the thigh. A better result might have been obtained had a solid nail been used in the supracondylar region and had it been possible to follow effects of traction with the X-rays. It seemed unwise to use more than 20 pounds' weight with traction on the head of the tibia.



FIG. 23.—Case IV. Front view taken 2 months after Fig. 21.



FIG. 24.—Case V. Total absorption of femoral neck 3 weeks after fracture in a syphilitic subject. The head lies against the shaft.

CASES IN WHICH NAIL-EXTENSION WAS APPLIED AND WHICH DIED FROM CAUSES HAVING NO RELATION TO THE METHOD EMPLOYED

CASE V. Alice K., aged fifty-two years (Dr. Collins' service, City Hospital).

September 13, 1912. Patient broke her right hip.

September 20. Supracondylar insertion of Steinmann's two-piece nail by Dr. H. H. Janeway.

September 21. Wassermann reaction was reported strongly positive. On account of her debilitated condition, she bore antispecific treatment poorly.

October 7. Seventeenth day after insertion, local pain around the nail; temperature, 102 degrees.

October 10. Twentieth day, fever having continued, nail removed; 2 drams of pus escaped; nail was found broken at the screw-joint; but the ends of the tongs had kept the pieces of nail in position at approximately right angles to the bone.

October 11. Temperature normal; discharge less.

October 15. No union. X-rays show total absorption of the femoral neck, so that head rests against shaft (Fig. 24).

October 18. The patient has been gradually declining for the past two weeks. During the past week there has been no fever; she became much weaker this day; went into coma, and died. Diagnosis: Death from syphilitic myocarditis. No autopsy.

Epicrisis. This was a fracture of the neck of the femur in a markedly syphilitic subject. Total absorption of the neck in thirty-two days. It is possible there was a previously existing gumma of the neck of the femur, predisposing to fracture at that point. The patient was intractably filthy in her habits. Inflammation of the nail hole, which occurred on the seventeenth day after insertion, promptly subsided after removal of nail and application of wet dressing. Patient died of her myocarditis a week after her temperature had reached normal. The nail broke at its joint under no undue strain. (Traction between 10 and 15 pounds.)

CASE VI. Fritz D., aged forty-eight (Dr. Collins' service, City Hospital).

October 27, 1912. A confirmed drunkard, fractured his thigh while intoxicated. He had delirium tremens four days after admission. Moderate icterus. Condition gradually improved.

November 1. Two-piece nail inserted; no anesthesia; 19 pounds' traction.

November 2. Comfortable.

November 3. Again developed delirium; irrational. Developed a pneumonia, to which he promptly succumbed.

November 5. X-rays, taken while in delirium, show broken nail held in place by tongs (Fig. 25). No autopsy.



FIG. 25.—Case VI. A two-piece nail which broke at the joint. The pieces were held at approximately right angles by the tongs (which do not show in the picture) and so continued to exert traction.

CERTAIN ASPECTS OF THE OPERATIVE TREATMENT OF FRACTURES

A. *The Freeing of Fragments Preliminary to Operative Reduction.*¹

In descriptions of the open treatment of fractures, especially fractures of the femur, very little is said of that part of the operation which consumes the most time, namely, the freeing of the fractured ends. One usually sees a rapid exposure of the bone surfaces nearest the operator; after this, the speed of the operation diminishes, as the difficult and laborious freeing of the posterior surfaces goes on. From time to time, attempts at reduction are made, followed by further freeing of the soft parts. Finally, with a good deal of effort, reduction is accomplished, and position is maintained by bone-holding appliances; the rest is easy routine work.

It is well known that the longer a fracture remains unreduced the more contraction of the soft parts there is. In other words, recent fractures are much easier to reduce than those of some weeks' standing. In fractures of several months' duration, the soft parts may become so inelastic that reduction can be accomplished only by the use of such an undue force that rupture of important vascular and nervous structures is rendered likely. Under such circumstances, a certain amount of bone must be resected before there can be a reduction without dangerous tension upon the soft parts.

The two chief opponents to reduction in fairly recent fractures with nonunion (after two months) are, first, connective tissue deposited in the vicinity of the fracture—this includes not only callus, but also scar tissue of the soft parts lacerated at the time of fracture; second, the contracted aponeurotic insertions of the muscles along the *linea aspera*, which can be considered as one structure.

As said before, the freeing of the fragments of their soft part attachment on the side nearer the operator (anterior and lateral) is easy; while on the side away from the operator (posterior and mesial) it is difficult, because the fragments themselves lie in front of the operative field, and the soft parts crowd in from either side. One of the chief structures hindering reduction—namely, the aponeurotic insertion of the muscles along the *linea aspera*—lies in this posterior region which is so difficult of access. This region can readily be made accessible by the following method:

As soon as the anterior surfaces of the bony fragments are freed, an assistant continually makes an attempt to kink the limb²; the soft parts hindering this are divided by repeated small cuts against the bone on either

¹ Taken in part from my article in the *Annals of Surgery*, October, 1912, p. 643.

² The angle thus formed has its apex pointing anteriorly.

fragment. As the resisting structures are gradually severed, the two fragments form an angle which constantly becomes more and more acute. It is now possible to see the posterior aspect of both fragments, and to free the bone of its aponeurotic attachments along the linea aspera for an inch or more on either fragment. By cutting against the bone, injury to vessels and nerves can be avoided. The limb is now straightened out and reduction is attempted; if sufficient relaxation has not been gained, the fragments are again brought vertical and parallel and their ends pulled apart (Fig. 26), so that the freed aponeurotic attachment along the linea aspera joining them is exposed, put on tension, and divided. In doing this tenotomy, great care must be observed in order to avoid injury, either to the femoral vessels and long saphenous nerve in Hunter's canal (in fracture of middle third of the thigh), or to the sciatic nerve, for at certain points these structures lie just beyond the aponeurotic tissue which is being divided.

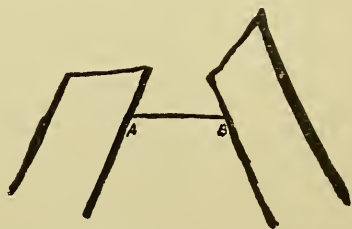


FIG. 26.—Diagrammatic representation of two fragments tilted anteriorly (upward); each has been freed of its muscular insertion (along the linea aspera) for an inch or so. The edge of the aponeurotic structure (AB) is shown joining the fragments.

To recapitulate: The aponeurotic insertions of the muscles along the linea aspera can be considered as one structure. It constitutes one of the chief obstacles to the reduction of a femoral fracture. A blind division of this structure (tenotomy), as it lies covered by the ends of the fractured bone, is a foolhardy procedure, because of possible damage to the sciatic nerve or to the femoral vessels, and because of the difficulty of controlling hemorrhage in such a situation. By gradually kinking the limb at the point of fracture, and dividing the soft parts which hinder this attempt, the posterior aspect of both fragments becomes readily accessible. Usually the freeing of both fragments of their soft part attachments (including tendons) for an inch or so suffices; but, if not, the linea aspera "tendons" can be exposed as before and a careful tenotomy be done.

The procedure is a simple, rapid, and safe method for freeing the ends of fragments. It removes a large proportion of the resistance to reduction offered by the soft parts. This method of exposure and division of tendinous structures opposing reduction is available in fractures of the other long bones which come to open treatment.

B. *The Reduction of Fragments by Distraction with Turnbuckles and Clamps.*³ If the fracture be transverse, the limb is kinked, the ends of the fragments are properly coapted, and the limb is then straightened out. The fragments stay in place.

If the fracture be oblique, greater difficulties are present, for the oblique surfaces slide past each other and the method just described is of no use.

³Taken in part from my articles in the *Annals of Surgery* of November, 1912, p. 769, and November, 1913, page 657.

Here, traction or distraction must be used to accomplish reduction, and must be maintained until the fragments are fixed by a suitable clamp or plate.

If the fracture be of the double oblique type (comminuted) (Fig. 27), still greater difficulties of reduction present themselves. The two main



FIG. 27.—Partially reduced double oblique fracture.



FIG. 28.—Completely reduced double oblique fracture.

fragments, when reduced, meet at what is practically a point (Fig. 28). Maintenance of such a position (until the plate or clamp has been applied) is very difficult.

In reducing a fracture, it is relatively easy to bring down the overlapping to within from one-half inch to one inch of complete reduction.

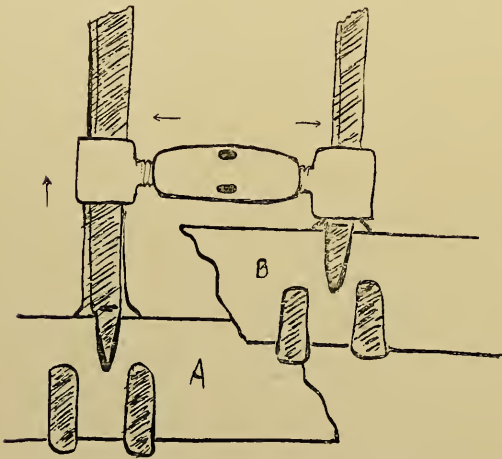


FIG. 29.—After sufficient distraction has been made, the shaft of the Lowman clamp holding fragment A can be pulled upward (sliding in the jaw of the turnbuckle, see arrow) so that the two fragments come into proper alignment. Just previous to alignment a Lane plate can be slipped under the clamp on fragment B. After alignment the plate is also slipped under the clamp on fragment A.

The nearer the complete reposition of fragments is approached, the greater is the resistance to reduction. In other words, it is the overcoming of this last inch or less of shortening which affords the greatest difficulty.

The obstacles encountered in operating on two fractures of the femur at the City Hospital (Blackwell's Island) in August, 1911, suggested to me that a simple effective method for reduction of fragments might be evolved which could be applied directly in the wound itself.

After proper exposure and freeing, a Lowman clamp (Figs. 29 and 30) is applied to each fragment. A turn-

turnbuckle (Figs. 30, 31a, and 31b) is so inserted that the jaws at its ends engage the shafts of the clamps on either fragment (Fig. 29).

As the barrel of the turnbuckle is rotated, the clamps (fragments) are forced apart. When the turnbuckle has reached its maximum extension, a second larger turnbuckle (Fig. 30) is applied and continues the extension as far as necessary.

The turnbuckle's jaws are made to *loosely* fit the shafts of the Lowman

clamps in order to permit of the fragments (which are out of line) being gradually brought into their proper relation.

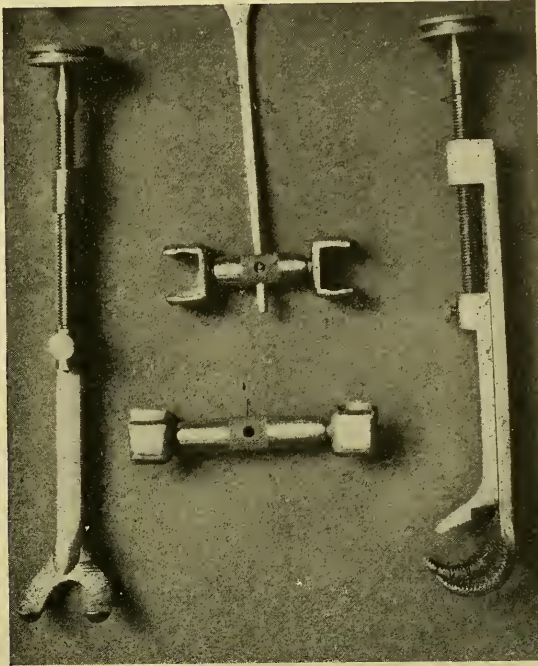


FIG. 30.—Anterior and lateral views of modified Lowman clamps. Smaller turnbuckle with key in place. Range of smaller turnbuckle is from $1\frac{7}{8}$ to $2\frac{1}{2}$ inches; range of larger turnbuckle is from $2\frac{3}{8}$ to $3\frac{1}{2}$ inches.

The following details of application must be kept in mind:

1. After both clamps are applied to the bone, and the turnbuckle is in place between them, one clamp should be loosened, but should be maintained

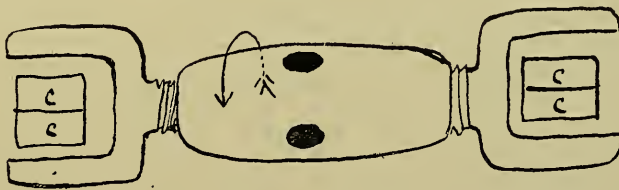
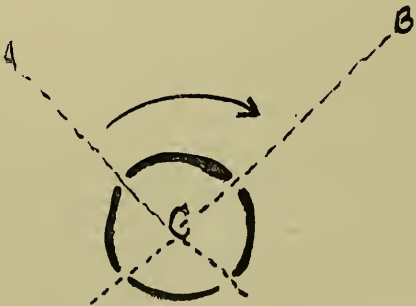


FIG. 31.—a) Turnbuckle. Jaws seen from above; they are purposely made large enough to fit the shafts of the Lowman clamps (cc) loosely, thus facilitating adjustment (alignment of fragments). b) Cross section of barrel of turnbuckle. One-quarter turn (by inserting pin into the hole and rotating) exposes the next hole. The right angle (A, C, B) defines the approximate amount of room afforded by the average wound.



in proper relation to the turnbuckle. Now the limb should be pulled upon, and when the maximum extension has been made, the clamp should be tightened and *extension is continued by means of the turnbuckle*. At times

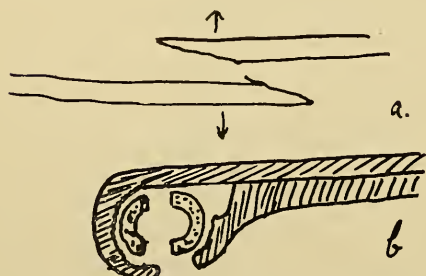


FIG. 32.—a) During distraction frequently there is a tendency to lateral displacement. b) A third Lowman clamp, which embraces both fragments but is not tight enough to prevent distraction (longitudinally), readily controls this.

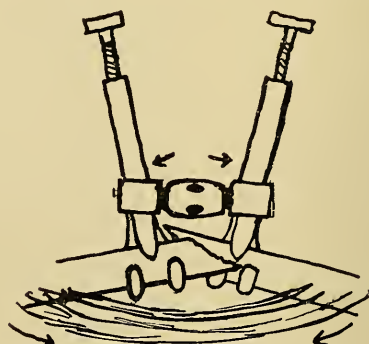


FIG. 33.—Showing tendency for distal ends of Lowman clamps to diverge because of resistance of soft parts.

lateral pressure may be necessary to keep the fragments in line during extension (Fig. 32).

2. As the turnbuckle forces the clamps apart, the resistance of the soft



FIG. 34.—Turnbuckle armed with hooks.

parts increases. This resistance is chiefly noticeable on the side of the bone *opposite* to where the turnbuckle is applied. The tendency is for the fragments to kink (Fig. 33), and for the shafts of the Lowman clamps to di-

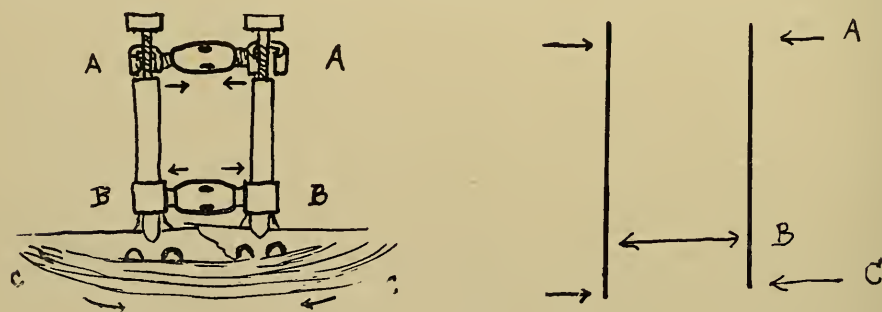


FIG. 35.—a) Divergence controlled by hooked turnbuckle at AA. Arrows indicate line of force. Usually it is more convenient to apply turnbuckle at BB, maximally contracted, and to apply hooked turnbuckle at AA, expanded sufficiently to engage diverging shafts. Then this same turnbuckle at AA is contracted until clamps are parallel or nearly so; after this, expansion of turnbuckle at BB can begin.

b) The Lowman clamps constitute levers; the turnbuckle at B is the fulcrum, the resisting soft parts at C, and the turnbuckle at A, balance each other.

verge instead of remaining parallel. If the divergence is at all marked, the turnbuckle's jaws slip up the shafts and slide out of place. To prevent this occurrence, it is necessary (a) *to place the turnbuckle as close down to the bone as possible*; (b) *to keep the two clamps parallel to one another by using a second turnbuckle armed with hooks* (Fig. 34). As the turnbuckle close to the bone forces the clamps apart, the upper turnbuckle is released just enough to maintain a proper parallel position of the Lowman clamps (Figs. 35a and 36).

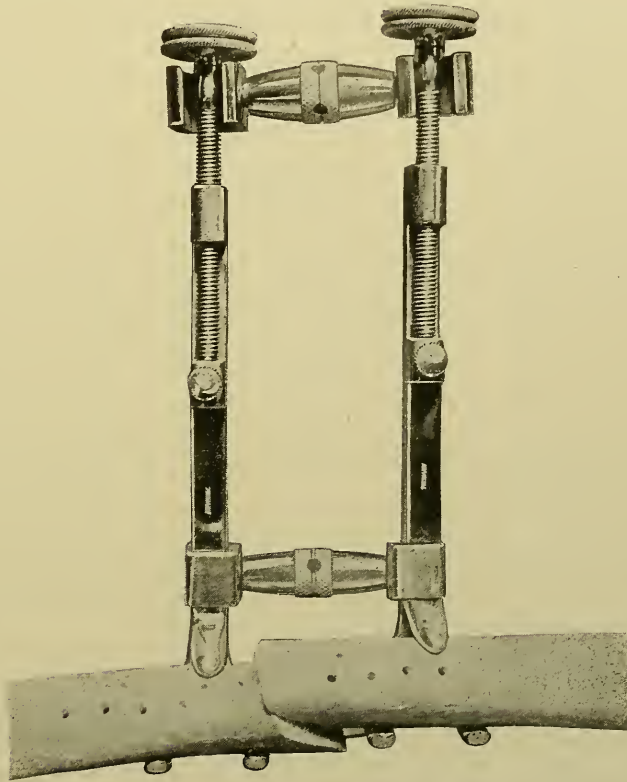


FIG. 36.—Photograph of clamps and turnbuckles applied to an adult femur as in Fig. 35.

Distention should be gradual, not sudden; after a certain amount of resistance is met with, it is well to pause a minute or two and then proceed again.

3. By applying the Lowman clamp so that the angular gap between it and the bone comes uppermost (Fig. 37), a Lane plate can be slipped along the bone without loosening the clamp or disturbing the turnbuckle. The plate may be placed beneath one clamp at any time before the fragments come in line. When proper alignment has been made, the plate is slipped through the gap in the clamp on the other fragment.

4. The Lowman clamps should be so applied that both their shafts lie to the same side of the bone, so as to facilitate proper alignment and to prevent turnbuckle and clamps from interfering with the drilling of holes for screws, etc.

5. In a double oblique fracture (Figs. 27 and 28), if the Lowman clamps be applied so that they partly overlap the oblique fractured surfaces of the main fragments, it will be possible, after slight hyperextension, to loosely

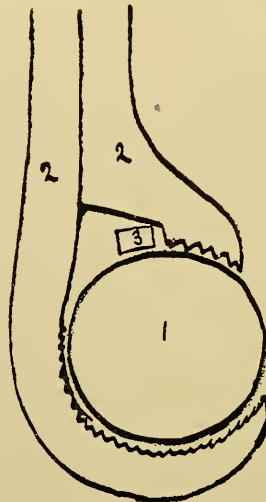


FIG. 37.—This diagram illustrates the heavier construction and slight modification of the Lowman clamp (2, 2) to permit convenient adjustment of the Lane plate (3) without disturbing the holder's grip upon the bone (1).

place the third triangular fragment into position, and then, by gradually allowing the main fragments to approach each other, the third fragment exactly occupies its proper location. The turnbuckle may even now be removed, previous to plating.

The turnbuckle and clamp method is applicable to any type of fracture in which the bone itself is sound. The method is simple, effective, and inexpensive. There is one danger attending its use, namely, the employment of too much power, with the possible rupture of important structures.

OF WHAT DIAGNOSTIC IMPORTANCE IS PAIN WHEN
REFERRED TO THE EYE?

BY

PERRY B. HOUGH, B.S., M.D.,
ASSISTANT ATTENDING OPHTHALMOLOGIST

OF WHAT DIAGNOSTIC IMPORTANCE IS PAIN WHEN REFERRED TO THE EYE?

BY PERRY B. HOUGH, B.S., M.D.,

Assistant Attending Ophthalmologist

We have been led to discuss the subject of pain as referred to the eye because we believe that very often the eye is subject to reflex pain caused by inflammatory conditions elsewhere. I believe we should divide our subject into two parts: First, those conditions of pain in the eye, accompanied by evidence of inflammation in the eye; and, second, those complaining of ocular pain without inflammatory symptoms. Under the first heading, we have all of the inflammatory conditions affecting the cornea, sclera, iris, and ciliary body. Pain is a constant factor in all affections of the cornea. A familiar example is foreign body of the cornea. In addition, we have the injection at the corneoscleral margin. This varies with the degree of inflammation; in foreign body of cornea it is often limited to a leash of blood-vessels in the same plane as the body is located and often serves as a guide to its presence. Where it has become infected, the congestion often surrounds the whole cornea. Very often patients come complaining of a sharp pain in the eye as of a foreign body, but examination of the cornea does not show any injection of it, but the eye has a watery appearance. Here, if we invert the upper lid, we will usually find a foreign body adhering to the conjunctiva. Sometimes we find instead simply a shred of mucus from a beginning conjunctivitis which will often give the same sensation. Ulcers of the cornea are always accompanied by severe pain, but examination of the cornea will show the broken surface. In scleritis we usually have pain more or less severe. The accompanying congestion of the scleral veins with the overlying conjunctiva gives the appearance so characteristic of scleral conditions as to render the diagnosis easy.

In iritis, in the beginning we have a sharp, stinging pain referred to the eye but soon referred to the temple. One characteristic of the pain is that it is usually worse at night. Pain, loss of motility of the iris, small pupil, and loss of markings with a change in the color of the iris, give us the diagnosis. In cyclitis, very often we have the iris involved as well, so the symptoms run together. In addition to the symptoms given for iritis, we have the distinctive points of local tenderness over the inflamed ciliary body, a cloudy cornea due to a deposit on Descemet's membrane, and a minus tension, the eye feeling soft.

In glaucoma, on the other hand, the eye becomes very hard; there is often a dull ache in the eye, but more often the pain is neuralgic in character and referred to the teeth or side of the head. The pupil is dilated and often has a greenish appearance. The attack quite often begins with vomiting, the patient thinking it a bilious attack.

Diseases of the conjunctiva as a rule do not cause pain unless complicated with a keratitis or iritis.

Abscess of the lid often causes severe pain, but the other signs of inflammation present serve to point to the diagnosis.

Chalazion, when undergoing suppuration, will often give a dull ache referred to the eye and orbit, but the swelling of the lid and redness leave no doubt as to the diagnosis.

Pain referred to the eye without inflammatory symptoms is much harder to diagnose, and usually a complete examination of the refraction and muscles is necessary before making a diagnosis. Then we must be careful in giving our prognosis as to the relief of pain from wearing glasses or muscle treatment. This side of the question can be given best by citing the histories of a few cases:

Dr. D. E. M., dentist, age 54, presented himself complaining of pain in the right eye, especially for near work. Examination showed that he was near-sighted, with near-sighted astigmatism in his right eye. His left eye had a combination of near and far-sighted astigmatism. His glasses he had been wearing were nearly correct. Further examination revealed a weakness of the muscles of convergence, and under appropriate treatment for this his symptoms disappeared. Six months later he returned, complaining of severe pain in the right eye, so severe his brother, a physician, had given him morphine the night before to relieve his pain. The eye showed no signs of inflammatory trouble, and believing sudden pain coming on without other signs is not due to refraction or muscle trouble, we had him transilluminated, which showed the right antrum dark. The antrum was punctured and washed out once, and a dead tooth extracted on that side. He has had no further trouble in the past three years.

CASE II. Dr. W. N. S.; age, 34; presented himself with the following history: Complains of attacks of severe pain in the eye followed by a blinding headache which incapacitates him for the day. First attack came on eight years ago, and would have three or four a week. Now only once in about ten days. Has been refracted several times, and has had muscles treated. Was advised to have a partial tenotomy of the external recti performed.

Examination of the vertical muscles of the eyes showed a difference in the level of one degree. The horizontal muscles seemed to be normal. Only a slight change in the glasses was found, the total changes not being thought sufficient to account for the symptoms. Examination of the nose showed large cystic middle turbinates with a deviated septum. The sinuses were negative. Turbinectomy was advised and a submucous resection of the

cartilage. As the right eye was subject to attack more often, turbinectomy was done on that side. Six months have elapsed since the operation, and he has had only two or three slight attacks, which I believe will totally cease when he has had the other side operated upon.

CASE III. Mr. C. L. H.; age, 35; came to see me complaining of a great deal of pain in his right eye and side of the head. Has had to wear dark glasses for the past few days. He also gave a history of having had trouble with his nose for some time. Examination showed vision normal, with a low degree of hyperopia. There were no signs of congestion of the eye, and the tension was normal. Examination of the nose showed it full of polypi. Shrinking with adrenalin chloride relieved some of the pain, and removal of the polypi cleared up his eye symptoms.

CASE IV. Mrs. S. L.; age, 32; came complaining of pain in the eyes all of the time. Has had trouble with her eyes for several years. Has been wearing glasses constantly for the past three years and now has a second pair for near work. Was operated upon for convergent strabismus when a child. Later the eye turned out and has been operated upon to correct that. Examination showed that she still had a divergence. Her astigmatism was not properly corrected, and her hyperopia was over-corrected. Proper correction of her refraction gave her marked relief, and correction and treatment of the muscles cleared up her trouble.

CASE V. Mr. W. W. H.; age, 34; was seen in consultation. Gave a history of trouble with eyes from early manhood. Had been unable to go to college because eyes would not permit him to study. If he read for a short time, eyes would pain him so that he was unable to use them again for the day. Had been refracted by several men without getting much benefit. Examination revealed a marked weakness of muscles of convergence. Under proper exercise and treatment, in about eight months symptoms completely disappeared, and he has had no return of them in the past eight years.

In conclusion: Sudden pain in the eye, accompanied with ocular congestion, usually denotes an affection of the cornea or uveal tract and is accompanied with other signs to make the diagnosis fairly simple. Sudden pain unaccompanied with ocular congestion is usually due to an inflammatory condition of the nose or accessory sinuses. Pain in the eyes of some weeks' duration, exaggerated by use of the eyes, may be due to refractive error or muscle imbalance, or very often a combination of both.

LIST OF GRADUATE INTERNS

LIST OF GRADUATE INTERNS

<i>Name</i>	<i>Address</i>	<i>Date of Graduation</i>
Abrams, A.	225 W. 110th Street, New York City	Oct., 1889
Adams, A. E.	44 Grand Street, Newburgh, N. Y.	Oct., 1880
Aldrich, John	164 W. 81st Street, New York City	Oct., 1892
*Allen, H. B.		Oct., 1880
Alsop, T.	New Brunswick, N. J.	June, 1897
Amabile, A. M.		Oct., 1867
Ambrose, D. R.		April, 1869
Ames, T. H.	52 W. 53rd Street, New York City	Dec., 1908
Anderson, F.		April, 1874
Andrews, J. L.	45 E. 92nd Street, New York City	Oct., 1890
Antony, C. L.	220 E. 123rd Street, New York City	Dec., 1901
Anway, J.	103 State Street, Chicago, Ill.	April, 1875
Arellano, G.	Granada, Nicaragua	Dec., 1899
*Asadoorian, A.		April, 1869
*Ashley, E. F.		June, 1904
Avery, O. T.	65 Central Park W., New York City	June, 1906
Bacon, H.	401 W. Duval Street, Jacksonville, Fla.	April, 1885
Bacon, W. F.	Hartford, Conn.	April, 1872
Baker, R. W.	1816 I Street, N. W., Washington, D. C.	April, 1889
*Baldwin, A. Van N.		Oct., 1883
Bancel, H. A.	235 E. 68th Street, New York City	June, 1911
Baner, W. L.	40 W. 68th Street, New York City	April, 1887
Banks, G.	Patterson, N. Y.	April, 1885
Barber, W. L.	87 W. Main Street, Waterbury, Conn.	June, 1909
Barker, H. L.	Woodside, N. Y.	April, 1894
Barrios, M. J.	Santa Rosa Siberia, Costa Rica	April, 1882
*Bartley, J. A.		Dec., 1901
Battle, K. P., Jr.	Raleigh, N. C.	Oct., 1883
Beardall, H. M.	Kansas City, Kans.	June, 1911
Beck, H. A.	Uhrichsville, Ohio	June, 1914
Becker, H. C.	229 W. 105th Street, New York City	Dec., 1905
Bellows, C. W.	433 Nostrand Avenue, Brooklyn, N. Y.	April, 1885
*Bertine, L. E.		April, 1885
Bettinger, J. W.	383 Endicott Arcade, St. Paul, Minn.	Oct., 1889
Billings, A. W.	Second Ave. & 74th St., Bay Ridge, Brooklyn.	Oct., 1888
*Birckhead, W. H.		April, 1865
Bissell, J. B.	46 W. 55th Street, New York City	Oct., 1884
Blackmar, B. G.	317 Ovington Avenue, Brooklyn, N. Y.	Oct., 1893
*Blake, J. A.		Oct., 1869
*Blondell, E.		Oct., 1879
Boise, Eugene.	Grand Rapids, Mich.	Oct., 1871
*Booth, W. H.		April, 1877
*Bosch, J. A.		April, 1887
Bowers, W. C.	336 State Street, Bridgeport, Conn.	Oct., 1879
Boyd, C. LeR.	Vincennes, Ind.	Dec., 1904
Boyd, C. S.	52 W. 84th Street, New York City	June, 1907
Boyd, W. A.	346 Willis Avenue, New York City	June, 1897
Boyde, A. S.	Panama, S. A.	April, 1901
Brace, H. M.	Perth Amboy, N. J.	April, 1883
Bradford, W.		April, 1867
Bradley, E. B.	172 N. Upper Street, Lexington, Ky.	Dec., 1905
Bradley, M. S.	South Manchester, Conn.	Oct., 1892
Brandt, W. J.	251 Carroll Street, Brooklyn, N. Y.	Oct., 1881
Breckwedel, H. B.	1849 Morgan Place, Los Angeles, Cal.	Aug., 1902

* Deceased.

<i>Name</i>	<i>Address</i>	<i>Date of Graduation</i>
Breese, A. B.	434 James Street, Syracuse, N. Y.	April, 1883
Bridges, A. C.	148 W. 74th Street, New York City.	Oct., 1885
Brill, W.	341½ Spring Street, Los Angeles, Cal.	April, 1889
Brinckman, A.	176 Bergen Street, Brooklyn, N. Y.	Oct., 1884
Brown, C. T.	117 N. Maple Avenue, East Orange, N. J.	Dec., 1908
Bryan, J. H.	818 17th Street, Washington, D. C.	Oct., 1879
Buck, H.	Toronto, Canada.	Dec., 1911
Buckley, E. W.	Ernst Building, St. Paul, Minn.	Oct., 1889
Buist, W. E.	Nashville, Tenn.	Dec., 1896
Bulkley, K.	531 Madison Avenue, New York City.	Sept., 1903
Bullock, T. S.	Lexington, Ky.	April, 1885
Bunnell, G. L.		Aug., 1902
Burdick, Carl.	717 E. John Street, Seattle, Wash.	June, 1913
*Burgess, M. G.		Aug., 1900
*Burns, J. F.		Oct., 1890
Burt, F.	Norwalk, Ohio.	April, 1880
Callan, P. A.	452 Fifth Avenue, New York City.	April, 1868
Carey, H. L. K.	222 E. 30th Street, New York City.	Dec., 1898
Carle, C. A.	Stanton, Mich.	Oct., 1867
Carpenter, F. B.	40 E. 41st Street, New York City.	Oct., 1882
Carpenter, G. B.	Detroit, Mich.	June, 1913
*Carpenter, H. T.		Oct., 1875
Carr, M. L.	353 W. 57th Street, New York City.	Oct., 1893
Carr, W. L.	68 W. 51st Street, New York City.	Oct., 1883
Carroll, J. H.	50 W. 9th Street, New York City.	June, 1910
Carson, J. B.	Farrel, Miss.	Oct., 1894
Carson, S. K.	Iloilo, P. I.	Aug., 1900
Carstens, C. F.	Keewatin, Itasco County, Minn.	Dec., 1909
*Carter, T. R.		April, 1892
Casamajor, L.	40 E. 41st Street, New York City.	Dec., 1907
*Castle, F. A.		April, 1867
Chapin, C. S.	Great Barrington, Mass.	Dec., 1897
Chapin, C. W.	Georgetown, N. Y.	Dec., 1904
Child, C. G., Jr.	163 E. 71st Street, New York City.	Aug., 1900
Cladek, W. E.	93 W. Milton Avenue, Rahway, N. J.	April, 1879
Clancy, W. H.	80 Otis Street, Cambridge, Mass.	Oct., 1887
Clark, A. N.	South Norwalk, Conn.	Oct., 1884
Clarke, H. M.	350 Park Avenue, Bridgeport, Conn.	Dec., 1910
Clarke, J. T.	United States Army.	Oct., 1889
Cleveland, C.	40 E. 41st Street, New York City.	Oct., 1871
Cocke, P. L.	Birmingham, Ala.	April, 1903
Cole, C. S.	122 W. 73rd Street, New York City.	April, 1889
Collyer, H. L.	153 W. 77th Street, New York City.	Oct., 1883
Conklin, J. H.	1 New Britton Avenue, Hartford, Conn.	Aug., 1902
Connolly, P. B.	United States Army.	Dec., 1910
Connors J. F.	224 W. 79th Street, New York City.	June, 1898
Cook, W. C.	733 Laurel Avenue, Cincinnati, Ohio.	Dec., 1906
Cornell, W. P.	217 Rutledge Avenue, Charleston, S. C.	Dec., 1901
Cornwall, L.	375 Park Avenue, New York City.	Dec., 1911
Corwin, B. F.	429 East 21st Street, Brooklyn, N. Y.	June, 1899
Corwin, T. W.	119 Belleville Avenue, Newark, N. J.	Oct., 1880
Coughlin, J. H.	63 E. 87th Street, New York City.	April, 1891
Cowherd, J. B.	Kansas City.	June, 1911
Cremin, P. W.	856 Lexington Avenue, New York City.	April, 1869
Croft, T. G.	Aiken, S. C.	June, 1914
*Crofton, J. R.		June, 1890
Croftutt, E. F.	Poultney, Vt.	June, 1906
Crowell, B. C.	Manila, P. I.	June, 1907
Crossman, L. W.	St. Luke's Hospital, Ponce, P. R.	Dec., 1912
Crane, R. W.	107 South St., Stamford, Conn.	June, 1907
*Culver, C.		Oct., 1868
*Culpepper, V. G.	Portsmouth, Va.	April, 1879
*Curtis, N. F.		April, 1875

* Deceased

<i>Name</i>	<i>Address</i>	<i>Date of Graduation</i>
Dakin, F. C.	1419 Chicago Avenue, Evanston, Ill.	Oct., 1892
Dalton, E. S.	694 E. 5th Street, Brooklyn, N. Y.	Dec., 1909
Dana, A. S.	Yonkers, N. Y.	Oct., 1874
Davis, G. P.	30 Woodland Street, Hartford, Conn.	April, 1870
Dawson, W. J. G. E.	California.	Oct., 1869
*Day, W. def.		Oct., 1864
Deady, H. B.		Dec., 1895
Delavan, D. B.	40 E. 41st Street, New York City	April, 1876
Deming, N. L.	Fort Wayne, Ind.	Oct., 1894
Denslow, LeG. N.		Oct., 1877
Depping, C. W.	Westfield, Mass.	June, 1914
Derr, E. Z.	United States Navy Yard, Boston, Mass.	Oct., 1872
Detwiller, A. K.	10 E. 58th Street, New York City	June, 1908
*Devan, S. C.		April, 1880
De Varona, J. C.	224 E. 31st Street, New York City	April, 1865
*Dew, J. H.		Oct., 1869
Dickson, J. R.	Waco, Texas.	Dec., 1911
Dillon, C. J.	161 W. 77th Street, New York City	June, 1904
Dinnan, J. B.	706 Howard Avenue, New Haven, Conn.	Dec., 1905
Donohue, G. H.	Northport, Long Island, N. Y.	Oct., 1884
Donovan, W.	131 E. 92nd Street, New York City	Aug., 1900
*Drake, F. R. S.		Oct., 1871
Drake, N. H.	United States Navy	Oct., 1873
Draper, J. W. (See Maury)		
Dudley, C. R.	4612 Finney Avenue, St. Louis, Mo.	Oct., 1893
Dunlop, S. R.	Mount Morris, N. Y.	Oct., 1891
Dunn, J.	Richmond, Va.	April, 1888
Dunn, W. W.	Richmond, Va.	June, 1896
*Dunne, R. C.		Oct., 1881
Dyas, T. C.	124 W. 114th Street, New York City	Dec., 1912
Earl, W. P.	654 E. Main Street, Little Falls, N. Y.	Aug., 1900
Eastman, A. F.	205 W. 8th Street, Erie, Pa.	Dec., 1909
Ecker, L. C.	1633 Connecticut Avenue, Washington, D. C.	June, 1908
Edgerton, F. D.	Middletown, Conn.	April, 1865
Edwards, L. B.	Richmond, Va.	April, 1868
Egan, P. R.	United States Army	Oct., 1881
*Embree, R. T.		Oct., 1885
Eno, H. C.	Saugatuck, Conn.	Oct., 1864
Estabrook, C. R.	9307 Flatlands Avenue, Brooklyn, N. Y.	Oct., 1877
Evans, A. P.	571 Park Avenue, New York City	Dec., 1907
*Everett, E. D.		Oct., 1864
*Farnham, A. B.	Milwaukee, Wis.	April, 1880
Farrell, E.	Nova Scotia.	April, 1865
*Farwell, G. D.		Oct., 1890
Feinberg, I. S.	310 E. 79th Street, New York City	Oct., 1890
Ferguson, B.		Dec., 1897
Ferguson, Robt.	Bisbee, Ariz.	Dec., 1904
Ferrer, J. M.	24 E. 60th Street, New York City	Oct., 1880
Finch, L. H.	188 Market Street, Amsterdam, N. Y.	June, 1905
*Fiset, G. O.		Oct., 1873
Fish, E. G.	510 N. Tioga Street, Ithaca, N. Y.	June, 1906
Fiske, J. P.	123 E. 53d Street, New York City	Oct., 1892
Fitzpatrick, C. B.	128 E. 93d Street, New York City	April, 1892
Fletcher, N. D. L.	556 W. 113th Street, New York City	June, 1904
Flores, R. deJ.	Costa Rica	Oct., 1871
Foote, S. K.	126 W. 103d Street, New York City	April, 1901
Ford, A. W.	244 Clinton Street, Brooklyn, N. Y.	April, 1873
Ford, C. M.	523 W. 141st Street, New York City	Oct., 1889
Ford, W. E.	266 Genesee Street, Utica, N. Y.	Oct., 1873
*Fortune, C. A.		Oct., 1872
Poster, T. G.	United States Navy	April, 1903
Frank, S. G.	362 Riverside Drive, New York City	April, 1895
Fraser, R.	Saranac Lake, New York City	Dec., 1898

* Deceased

<i>Name</i>	<i>Address</i>	<i>Date of Graduation</i>
Freeland, J.		Dec., 1896
French, C. H.	Pawtucket, R. I.	April, 1882
*Friedenberg, A.		Oct., 1879
Fullilove, T. W.	Vaiden, Miss.	April, 1876
Galloway, G. E.	Rahway, N. J.	Dec., 1899
Gardiner, C. F.	Colorado Springs, Colo.	Oct., 1883
*Garnett, A. P.		April, 1884
Garside, C. Z.	Fordham Univ., Sch. of Med., New York City	June, 1905
Garvin, A. H.	Raybrook Sanatorium, N. Y.	June, 1905
Garvin, Wm. C.	Manhattan State Hospital, Wards Island, N. Y.	Dec., 1904
Gaston, J. L.	Montgomery, Ala.	Oct., 1886
Gaunt, T. T.	53 W. 50th Street, New York City	April, 1882
Giffen, R. E.	Lincoln, Neb.	April, 1882
Gilday, A. L. C.	7 Seymour Avenue, Montreal, Can.	Sept., 1903
Giles, R.	Cold Spring, N. Y.	April, 1884
Gilham, C.	Charlottesville, Va.	April, 1890
Gillan, J. J.		June, 1896
Gilley, W. C.	176 W. 81st Street, New York City	April, 1887
Glover, F. R.	Mount Vernon, N. Y.	Oct., 1886
Goodall, H. S.	Bennington, Vt.	April, 1892
Goodwin, C. S.	527 State Street, Bridgeport, Conn.	Dec., 1897
Gorton, O. A.	Sherburne, N. Y.	April, 1878
Gottheil, W. S.	154 W. 77th Street, New York City	April, 1883
Grafstrom, A. V.	Jamestown, N. Y.	June, 1897
Grandy, C. R.	Norfolk, Va.	April, 1894
Grant, J. J.	Freeport, Ill.	June, 1913
Grant, T. T.		April, 1882
Graves, S. C.	3603 Lindell Boulevard, St. Louis, Mo.	Oct., 1884
Gray, G. C.	Edmonton, Alberta, Canada	Oct., 1868
Green, E. H.	United States Navy	April, 1874
*Greenway, A. W.		April, 1894
Grice, J.	Portsmouth, Va.	April, 1895
Griffin, W. W.	226 W. 50th Street, New York City	Dec., 1910
Groeshal, L. B.	47 Ft. Washington Avenue, New York City	June, 1908
Guillin, M.	Granada, Nicaragua	Dec., 1901
Gülshon, J. J.		Dec., 1899
Guinlock, W. C.	Seaforth, Ont.	Oct., 1868
Guiteras, R.	80 Madison Avenue, New York City	April, 1887
Guthrie, W. G.	Marysville, Kans.	June, 1912
Haddad, R. G.	56 Garden Place, Brooklyn, N. Y.	April, 1903
Hale, F.	477 State Street, Bridgeport, Conn.	June, 1911
Hall, D. M.	5 Pine Street, Glen Falls, N. Y.	Dec., 1895
Hamilton, W. L.		April, 1882
Hamlin, G. D.	1259 Pacific Street, Brooklyn, N. Y.	Oct., 1884
Haney, J. P.	1233 Third Avenue, New York City	Oct., 1893
Hardin, B. L.	1132 Connecticut Avenue, Washington, D. C.	June, 1897
*Harrington, DeW.		Oct., 1867
Harris, G. R.	Marshalltown, Iowa	April, 1888
Harrison, G.	204 W. 70th Street, New York City	April, 1892
Harrison, H. W.	Roanoke, Va.	Oct., 1886
Harrison, S. D.	408 N. Main St., Elmira, N. Y.	Oct., 1880
Harrison, W. H.		April, 1865
Hawes, W. A.		Oct., 1867
*Hazen, A.		Dec., 1896
Hazen, H. C.	66 W. 56th Street, New York City	Oct., 1892
Healy, W. P.	535 Park Avenue, New York City	Aug., 1902
Heger, A.		April, 1895
Heller, T. F.	504 W. 112th Street, New York City	April, 1885
Henschel, J. J.	288 E. 161st Street	Dec., 1895
Herbig, F. J.	College Point, L. I.	June, 1913
Hewett, W. B.	227 Clermont Avenue, Brooklyn, N. Y.	April, 1879
Heydecker, H. R.		April, 1891
Higgins, S. L.	United States Navy	June, 1905

* Deceased.

<i>Name</i>	<i>Address</i>	<i>Date of Graduation</i>
Higley, H. A.	184 Joralemon Street, Brooklyn, N. Y.	April, 1890
Hill, C. E.	Toronto, Canada	June, 1910
Hill, E. W.	600 Doan Street, Glenville, Ohio	Oct., 1875
Hill, E. Y.	320 W. 87th Street, New York City	Oct., 1890
Hillyer, J. H.		April, 1878
Hitchcock, DeW.	Oxford, N. Y.	April, 1878
Hitchcock, U. G.		April, 1870
Hodgson, J. H. P.	29 Washington Square, New York City	Oct., 1890
Hoffman, O.	566 S. 38th Street, Omaha, Neb.	April, 1877
Holladay, G. G.	Portsmouth, Va.	April, 1895
Holloway, J. H.	United States Navy	Dec., 1898
*Holmes, W. C.		April, 1881
Holmes, J. F.	1165 Boston Road, Bronx, N. Y.	1877
*Holstein, G. D.		Oct., 1883
Hough, P. B.	40 E. 41st Street, New York City	April, 1901
Howard, C. N.	1936 Cincinnati Street, Washington, D. C.	Aug., 1902
Hoyt, Randal	375 Park Avenue, New York City	June, 1912
Hoyt, W. G.	Stamford, Conn.	Oct., 1883
Huber, J. B.	40 E. 41st Street, New York City	April, 1891
Hughes, H.	Long Branch, N. J.	April, 1875
*Hughes, J. L.	21 Archer Avenue, Mount Vernon, N. Y.	Dec., 1901
Hull, M. McH.	Atlanta, Ga.	June, 1897
*Huntington, H. R.		Oct., 1872
*Hustace, F.		April, 1875
Hutchinson, H. S.	15 Grand Boulevard, Binghamton, N. Y.	Oct., 1894
Inches, C.	Scribner, Neb.	April, 1896
Inches, P.		Oct., 1866
Irwin, J. S.	Danville, Va.	April, 1895
Ives, F. J.	United States Army	Oct., 1880
*Janeway, E. G.		Oct., 1864
Jarecky, H.	138 W. 86th Street, New York City	April, 1888
Jayne, W. A.	Denver, Colo.	Oct., 1875
Jewett, C. R.	1299 Maine Street, Buffalo, N. Y.	Oct., 1882
Jewett, C. S.	892 Main Street, Buffalo, N. Y.	Oct., 1891
*Jobs, N. C.		Oct., 1875
Johnson, H. D.	The Belnord, B'way & 86th Street, N. Y. C.	June, 1899
Johnson, R. W.	United States Army	April, 1881
Johnson, W. B.	170 Broadway, Paterson, N. J.	April, 1880
Johnston, A. M.	89 Kossuth Avenue, Utica, N. Y.	June, 1898
Judd, H. B.	14 Central Park, West, New York City	June, 1906
Judson, E. U.		Oct., 1880
Kane, A. M.	32 W. 48th Street, New York City	April, 1894
Kaprelian, H. K.	66 South Street, Stamford, Conn.	June, 1910
Katzenberg, M.	1315 Madison Avenue, New York City	Oct., 1887
Kellogg, W. V.		June, 1896
Kelly, H. T.	White Plains, N. Y.	Aug., 1900
Kennedy, J. B.	Church St., Richmond Hill, Long Island, N. Y.	Oct., 1887
*Kennedy, J. M.		April, 1889
Kessler, G. L.	588 Bedford Avenue, Brooklyn, N. Y.	Dec., 1898
*Kimball, D.		Aug., 1900
Kinney, Alfred C.	259 Commercial Street, Astoria, Oregon	April, 1872
Kinney, Augustus C.	Astoria, Oregon	Oct., 1871
Klotz, W. C.	Los Angeles, Cal.	Dec., 1899
Knickerbocker, G. S.	145 W. 128th Street, New York City	Oct., 1876
Knight, W. W.	254 Trumbull Street, Hartford, Conn.	Oct., 1876
Kohn, A.	122 E. 58th Street, New York City	April, 1893
Kohn, S.	13 E. 75th Street, New York City	April, 1878
Krans, E. S.	920 Park Avenue, Plainfield, N. J.	June, 1909
Landsman, S. M.	220 E. 19th Street, New York City	Oct., 1890
Larew, J. J.	1110 N. Carson Avenue, St. Louis, Mo.	April, 1876
Laws, H. J.	Crown Point, Ind.	June, 1909
Lee, C. R.	Fulton, N. Y.	April, 1878
*Lee, W.		April, 1865

* Deceased.

<i>Name</i>	<i>Address</i>	<i>Date of Graduation</i>
Leipziger, H. A.....	Burlington, Iowa.....	April, 1884
Lenev, J. M.....	Winnipeg, Canada.....	Sept., 1903
*Leon, A. M.....		Oct., 1879
Leonard, C. H.....	154 Broad Street, Providence, R. I.....	Oct., 1869
*Levy, H.....		Oct., 1879
Likely, D. S.....	44 W. 96th Street, New York City.....	Dec., 1907
Lindsay, M.....	Topeka, Kans.....	Dec., 1911
*Linehan, J. B.....		Oct., 1876
Lippman, C. W.....	Hotel Richelieu, San Francisco, Cal.....	June, 1912
Little, H. W.....	Evansville, Ind.....	April, 1903
Livingston, E. P.....	808 St. Nicholas Avenue, New York City.....	Dec., 1898
Lockwood, W. A.....		Oct., 1864
Ludbrook, W. W.....	2070 Fifth Avenue, New York City.....	Dec., 1896
Lydston, G. F.....	100 State Street, Chicago, Ill.....	April, 1881
Lyle, A.....	1043 Madison Avenue, New York City.....	April, 1890
Lynch, C. F.....	Springfield, Mass.....	Dec., 1898
Lynch, L. C.....	919 St. John's Place, Brooklyn, N. Y.....	Dec., 1905
Lynde, N. W.....	51 W. 91st Street, New York City.....	April, 1887
*Lyon, E. R.....		Oct., 1886
*Lyttle, H. T.....		April, 1879
McAuliffe, G. B.....	616 Madison Avenue, New York City.....	Oct., 1889
McBride, J. H.....	Pasadena, Cal.....	April, 1874
McCabe, J.....	348 Central Park West, New York City.....	June, 1904
McCafferty, J.....	36 W. 59th Street, New York City.....	April, 1901
McCarthy, P. F.....	Marlborough, Mass.....	Oct., 1892
McCartney, G.....	Toronto, Canada.....	Sept., 1903
McChesney, J. Q.....	Plumas County, Cal.....	Oct., 1882
McDermott, J.....	204 W. 85th Street, New York City.....	April, 1895
McEwen, J. S.....	Orlando, Fla.....	Dec., 1906
McGay, R. J.....		Oct., 1868
McGill, E. L.....	Petersburg, Va.....	Sept., 1903
McKeller, J. M.....	Tenaflly, N. J.....	June, 1907
McKinley, N. J.....	Toronto, Canada.....	June, 1910
McKinney, J. A.....	Lancaster, Ont.....	Dec., 1908
McLennon, A. L.....		April, 1873
McMannis, W. T.....	612 W. 179th Street, New York City.....	April, 1887
McNamara, T. L.....	Corning, N. Y.....	Dec., 1912
Macomb, E.....		Oct., 1866
Maghee, J. M.....	Orange, N. J.....	Oct., 1888
*Malleon, P. H.....		April, 1886
Mallett, E. P.....	404 Bryant Building, Kansas City, Mo.....	Oct., 1891
Mallett, G. H.....	244 W. 73rd Street, New York City.....	April, 1889
Manges, M.....	72 E. 79th Street, New York City.....	Oct., 1888
Manison, W. H.....		April, 1894
Mann, Baldwin.....	146 E. 71st Street, New York City.....	Dec., 1910
Mann, J.....	Petersburg, Va.....	June, 1897
Manning, A.....	70 Park Avenue, Plainfield, N. J.....	Oct., 1885
Marcus, L.....	1215 Madison Avenue, New York City.....	June, 1899
*Marston, D. W.....		Aug., 1900
Martland, H. S.....	1138 Broad Street, Newark, N. J.....	June, 1907
*Marx, S.....		Oct., 1885
Matthews, F. C.....	446 Central Park West, New York City.....	June, 1904
Maupin, E. G.....	151 E. 127th Street, New York City.....	April, 1880
Mauzy, J. W. Draper.....	104 E. 40th Street, New York City.....	Dec., 1899
*Maxwell, E. A.....		April, 1870
Mayer, A.....	40 E. 60th Street, New York City.....	Oct., 1876
*Meade, J. C.....		April, 1869
Meeker, H. D.....	220 W. 79th Street, New York City.....	June, 1904
Meirowitz, P.....	530 Manhattan Avenue, New York City.....	Oct., 1891
*Merriam, F.....		April, 1881
Merrigan, T. D.....	553 W. 167th Street, New York City.....	April, 1889
Meyer, A. E.....		Oct., 1894
Meyer, J.....	216 Van Buren Street, Brooklyn, N. Y.....	April, 1878

* Deceased

<i>Name</i>	<i>Address</i>	<i>Date of Graduation</i>
Meyer, L. L.	Masonic Temple, Memphis, Tenn.	June, 1898
*Meyers, J.		July, 1871
Michaelis, L. M.		April, 1890
Miller, A. I.	Brattleboro, Vt.	April, 1885
Miller, G. E.	2804 Glenwood Road, Brooklyn, N. Y.	June, 1909
Milroy, W. F.	312 McCagne Building, Omaha, Neb.	Oct., 1884
Mitchell, C. R.	16 Church Street, Paterson, N. J.	June, 1908
Mixsell, R. B.	Pasadena, Cal.	June, 1909
Moore, A. A.	121 Madison Avenue, New York City.	June, 1898
Moore, B. W.	3634 Washington Avenue, St. Louis, Mo.	Dec., 1896
Moore, W.		April, 1886
Moore, W. O.	616 Madison Avenue, New York City.	Oct., 1873
Morales, R.	Costa Rica.	Oct., 1871
Morrison, R. L.	14 Wilton Crescent, Toronto, Canada.	June, 1913
Morrison, W. H.	167 W. 91st Street, New York City.	April, 1894
Mossman, J. K.		Dec., 1912
Munson, A. E.	2387 Davidson Avenue, New York City.	April, 1901
Murphy, A. J.	463 E. 1st South Street, Salt Lake City, Utah.	June, 1908
Murray, E. W.	91 Washington Avenue, Newark, N. J.	Dec., 1899
Murtland, S.	40 E. 81st Street, New York City.	Oct., 1884
*Muzzy, A. T.		Oct., 1881
Myers, H. G.	153 W. 78th Street, New York City.	Oct., 1888
Myron, H.	Cor. Dudley & Putnam sts., Syracuse, N. Y. .	June, 1910
Neff, J. H.	Harrisonburg, Va.	Oct., 1872
Newburgh, J. L.	4500 Vincennes Avenue, Chicago, Ill.	Oct., 1886
Newman, A. M.		April, 1891
Newsom, T. C.	Independence, Miss.	Dec., 1913
Newton, R. C.	42 Church Street, Montclair, N. J.	April, 1879
*Nolan, F. W.		June, 1899
Norfleet, E.	Roxobel, N. C.	April, 1874
Nutt, C. W.		April, 1874
O'Brien, D.		Dec., 1901
O'Brien, M. C.	161 W. 122nd Street, New York City.	April, 1882
O'Brien, P.	Carlstadt, N. J.	Dec., 1909
*O'Dwyer, J.		April, 1868
O'Dwyer, J.	78 E. 56th Street, New York City.	Aug., 1902
Ogilvy, C.	40 E. 41st Street, New York City.	April, 1901
Old, H.	Norfolk, Va.	June, 1897
Oppenheim, N.	50 E. 79th Street, New York City.	Oct., 1892
Oppenheimer, H. S.	11 E. 43rd Street, New York City.	Oct., 1877
Ordway, W. H.	591 Park Avenue, New York City.	June, 1914
Osborne, J. D.	Petersburg, Va.	June, 1898
*Ottley, C. W.		Aug., 1902
Overton, F.	Patchogue, Long Island, N. Y.	Oct., 1894
Owens, F. T.	99 Charlotte Street, Utica, N. Y.	Dec., 1908
Paine, A. G.	3964 Drexel Building, Chicago, Ill.	April, 1878
*Pardee, E. L.		April, 1872
Park, C. R.	745 Quincy Avenue, Scranton, Pa.	Oct., 1885
*Parsons, F.		Oct., 1877
Partridge, E. L.	19 Fifth Avenue, New York City.	Oct., 1874
Patterson, W. M.	354 W. 21st Street, New York City.	Dec., 1913
*Pease, D. P.		April, 1884
*Pell, R. V.		Oct., 1866
Pemberthy, G. C.	145 King Avenue, Detroit, Mich.	Dec., 1911
Peters, H. LeB.	467 State Street, Bridgeport, Conn.	June, 1909
Percival, F. R.	2635 W. Pico Street, Los Angeles, Cal.	April, 1889
Pereira, B.	Mompos, United States of Colombia.	April, 1880
Phelps, S.	Malone, N. Y.	Aug., 1900
Pick, R. E.	230 E. 50th Street, New York City.	June, 1905
Pierce, A. M.	New Bedford, Mass.	Oct., 1874
Pierson, S.	Stamford, Conn.	April, 1883
*Piffard, H. G.		Oct., 1864
Pike, A. S.	165 Park Avenue, Rochester, N. Y.	June, 1898

* Deceased.

<i>Name</i>	<i>Address</i>	<i>Date of Graduation</i>
*Pinto, V.		April, 1877
*Plimpton, W. O.		April, 1890
Polhemus, A. S.	United States Army	April, 1883
Pollock, L. W.	Temple, Texas	Dec., 1904
Pomeroy, J. L.	Norris & Stevens aves., Louisville, Ky.	June, 1905
Pomeroy, R. H.	511 Nostrand Avenue, Brooklyn, N. Y.	April, 1891
Potter, P. A.	26 Halstead Street, East Orange, N. J.	Dec., 1901
Potter, W. W.	Spokane, Wash.	Oct., 1872
Powers, W. Z.		Oct., 1879
Pratt, E.	Torrington, Conn.	Oct., 1888
Pratt, J. H.	Manchester, N. Y.	April, 1891
Price, Thos. I.	Central & Neurological Hosp., Bl'kw'll's Isl.	Dec., 1910
Priest, F. K.	Nashua, N. H.	April, 1883
Primrose, A. J.	89 West 103rd Street, New York City	April, 1888
Proben, C. I.	160 E. 71st Street, New York City	Oct., 1891
Province, O. A.	Indianapolis, Ind.	June, 1904
Provost, A. G.	New Haven, Conn.	Dec., 1906
Pumyea, P. C.	574 West End Avenue, New York City	Dec., 1906
Quimby, W. G.	14 James Street, Newark, N. J.	Aug., 1902
Quin, V. E.	889 Cauldwell Avenue, New York City	April, 1901
Quinlan, W. W.	103 State Street, Chicago, Ill.	April, 1893
Quinn, J. F.	103 W. 73rd Street, New York City	April, 1903
Quinton, W. W.	United States Army	Oct., 1893
Rafferty, O.	United States Army	April, 1886
Rassman, W. H.	71 Minor Avenue, Rutherford, N. J.	Oct., 1885
Raymond, T. N.		June, 1890
Reardon, W. I.	105 E. 53rd Street, New York City	June, 1913
Reid, R. W.	267 W. 114th Street, New York City	Dec., 1897
Reilly, T. F.	220 W. 139th Street, New York City	Dec., 1897
Remmer, H. T.	Gardiner Building, Utica, N. Y.	Dec., 1913
Reynolds, W. S.	132 W. 73d Street, New York City	Oct., 1893
*Rhea, J.		July, 1883
Rice, C. C.	123 E. 19th Street, New York City	April, 1879
Richard, C.	United States Army	Oct., 1877
Richmond, N. G.	Fredonia, N. Y.	Oct., 1881
Rinard, C. C.	317 Eighth Avenue, Homestead, Pa.	Dec., 1899
Roberts, S. M.	Siasconsett, Mass.	Oct., 1868
Robertson, J. L.		Oct., 1869
Robertson, W. M.	2608 Locust Street, St. Louis, Mo.	Oct., 1891
Rockwell, A. V.	931 E. 178th Street, New York City	June, 1898
Rockwell, T. H.	6 Princess Street, London, Eng.	Oct., 1888
Rodriguez, C. F.	Cuba	Oct., 1872
Rosenthal, A.	Farmington, N. M.	April, 1892
Ross, O. B.	702 W. Trade Street, Charlotte, N. C.	June, 1911
*Ross, W. H., Jr.		Oct., 1869
Roy, C. D.	Atlanta, Ga.	April, 1891
Rucavado, G.	San Jose, Costa Rica, Central America.	Oct., 1876
Ruffin, K.	Norfolk, Va.	April, 1888
*Rupp, A.		April, 1879
*Rush, C. W.		Oct., 1877
Russell, J. F.	239 Thompson Street, New York City	Oct., 1881
Saunders, N. B.	172 Stevens Avenue, Mt. Vernon, N. Y.	April, 1903
Schlierbach, T. L.	6558 Normal Avenue, Chicago, Ill.	April, 1887
Schoonover, W.	115 E. 59th Street, New York City	April, 1868
Schroeder, H. H.	32 Nassau Street, New York City	April, 1888
Schuyler, W. J.	3 Hopper Street, Utica, N. Y.	Oct., 1886
Scudder, C. F.	Northport, Long Island, N. Y.	June, 1914
Searle, D. W.		Oct., 1867
Sheard, C. J.	Toronto, Canada	Dec., 1911
Shears, G. P.	203 W. 103rd Street, New York City	April, 1895
Sheldon, Luther	United States Navy	Dec., 1910
Shellman, A. P.	457 Chenango Street, Binghamton, N. Y.	Dec., 1895
Sherman, A. L.	449 Park Avenue, New York City	June, 1906

* Deceased.

LIST OF GRADUATE INTERNS

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<i>Name</i>	<i>Address</i>	<i>Date of Graduation</i>
*Sherman, G. E.		Oct., 1868
Shrady, J. E.	151 W. 73d Street, New York City	April, 1894
Siglar, H. B.	24 State Street, New York City	June, 1904
Sill, E. M.	104 W. 76th Street, New York City	Aug., 1900
Slabaugh, W. H.	Omaha, Neb.	April, 1884
Slack, J. D.	1019 Ogden Avenue, New York City	Dec., 1906
Smillie, J. G.	Guelph, Ontario, Canada	April, 1903
Smith, A. H.	Camden, N. J.	Oct., 1877
Smith, D. H.	Bath, N. Y.	Oct., 1874
*Smith, E. L.		April, 1891
Smith, H. L.	Watertown, N. Y.	April, 1865
Smith, H. M.	20 Howell St., Canandaigua, N. Y.	Dec., 1913
Smith, W. E.	295 Atlantic Ave., Stamford, Ct.	June, 1912
Snyder, R. G.	375 Park Avenue, New York City	June, 1908
Somerset, W. L.	107 Lexington Avenue, New York City	Oct., 1893
Southack, J. W.		Oct., 1864
*Southworth, E.		Oct., 1882
Sparkman, Ed.	138 Broad Street, Charleston, S. C.	Dec., 1907
Sparkman, J. R.	Charleston, S. C.	Dec., 1909
Spencer, W. D.	Saybrook, Conn.	Oct., 1875
Spiller, W. H.	307 Second Avenue, New York City	June, 1897
Sprague, D. H.	Central Valley, N. Y.	April, 1888
Stark, O. W.	11½ 8th Street, Cincinnati Ohio	April, 1888
Stark, N. M.	55 W. 95th Street, New York City	Sept., 1903
*St. John, H.		Oct., 1887
Steers, T. H.	410 W. 43d Street, New York City	April, 1881
Steinach, W.	239 E. 52nd Street, New York City	Dec., 1895
*Stephenson, C.		Oct., 1891
Stewart, D. H.	128 W. 86th Street, New York City	April, 1884
Stewart, J. H.	425 Portland Avenue, St. Paul, Minn.	April, 1885
Stewart, W. H.	222 West 79th Street, New York City	April, 1893
Stewart, W. W.	Columbus, Ga.	Oct., 1891
Stieglitz, E.	42 W. 95th Street, New York City	April, 1886
Stimson, D. M.	28 W. 37th Street, New York City	Oct., 1868
Stone, W. R.	73 W. 49th Street, New York City	April, 1901
Story, G.	Portland, Oregon	Dec., 1895
Stowell, W. L.	160 W. 73rd Street, New York City	Oct., 1883
Sugg, J. P.	Kittrell, N. C.	Oct., 1868
Sullivan, J. D.	74 MacDonough Street, Brooklyn, N. Y.	April, 1868
Sullivan, J. F.	205 Blatchley Avenue, New Haven, Conn.	June, 1896
Sutton, F. A.	Plain Street, Hackettstown, N. J.	Dec., 1908
Swasey, J. H.		April, 1876
*Swift, L. C.		April, 1880
Sykes, R. L.	Columbus, Miss.	April, 1867
Symmers, D.	338 E. 26th Street, New York City	Dec., 1904
Taddiken, P. G.	Ogdensburg, N. Y.	Dec., 1896
Talbot, A.	203 Rialto Building, Kansas City, Mo.	April, 1866
Talmage, J. B.	1356 Prospect Avenue, New York City	Dec., 1905
Tanquary, R. J.	930 Belt Avenue, St. Louis, Mo.	Dec., 1912
Taylor, F. L.	112 E. 60th Street, New York City	April, 1893
Taylor, J. S.	United States Navy	June, 1896
Taylor, W. O.	San Francisco, Cal.	Oct., 1866
*Teakle, G. W.		April, 1872
Terriberry, J. F.	120 W. 73rd Street, New York City	April, 1882
Thelberg, M. A. H.	Riddergarten 35, Stockholm, Sweden	Dec., 1899
Thomas, M. D.	829 South Railroad Avenue, Opelika, Ala.	June, 1906
Thomas, R. H.	167 College, Street, Toronto, Canada	June, 1911
*Thompson, A. M.		April, 1893
*Thompson, C. N.		April, 1886
*Thomlinson, J.		April, 1879
Tobias, L.	326 E. 52nd Street, New York City	Dec., 1898
Toole, A. F.	Birmingham, Ala.	Sept., 1903
Torrens, B.	254 W. 102nd Street, New York City	April, 1895

* Deceased

<i>Name</i>	<i>Address</i>	<i>Date of Graduation</i>
Tousey, R.	903 Park Avenue, New York City	June, 1899
*Townsend, F., Jr.	McGrange, Cook County, Ill.	Oct., 1877
Trask, F.	McGrange, Cook County, Ill.	Oct., 1876
*Trask, H. P.	Yokohama, Japan	April, 1890
Tripler, T. H.	Yokohama, Japan	Oct., 1869
Tynberg, S.	1329 Madison Avenue, New York City	Oct., 1886
Uran, A. W.	2337 W. Harold Street, Philadelphia, Pa.	June, 1914
*Urquart, F. M.	St. Paul, Minn.	Oct., 1881
*Van Horne, A. B.	Washington, D. C.	Oct., 1881
Van Rensselaer, J.	Oakdale, Cal.	Oct., 1890
Van Winkle, O.	68 Pratt Street, Hartford, Conn.	April, 1889
Waite, F. L.	54 W. 71st Street, New York City	April, 1890
Waldo, R.	6 W. 83rd Street, New York City	April, 1883
Walker, D. E.	Summerville, Mass.	April, 1887
Walker, H. A.	120 E. Broad Street, Columbus, O.	Dec., 1906
Wardlow, Y.	4424 Drexel Boulevard, Chicago, Ill.	Oct., 1892
Ware, L.	St. Paul, Minn.	Oct., 1867
Warren, E. L.	St. Paul, Minn.	June, 1912
Watkins, W. B.	St. Paul, Minn.	June, 1898
*Waterman, J. H.	St. Paul, Minn.	Dec., 1895
Weiss, G. C.	154 Stevens Avenue, Mt. Vernon, N. Y.	April, 1884
Welch, H. L.	44 College Street, New Haven, Conn.	Dec., 1898
Welch, J. E.	138 W. 70th Street, New York City	April, 1903
Weldon, L. O.	Des Moines, Ia.	June, 1910
Wells, B. H.	523 Madison Avenue, New York City	Oct., 1885
Wells, F. C.	175 Belleville Avenue, Bloomfield, N. J.	Oct., 1888
Wells, F. L.	Des Moines, Ia.	Oct., 1889
*Wells, F. M.	40 E. 41st Street, New York City	April, 1881
Wells, J. G.	887 Park Place, Brooklyn, N. Y.	Dec., 1901
Wheeler, R. T.	325 Clinton Avenue, Newark, N. J.	Oct., 1893
Wherry, E. G.	325 Clinton Avenue, Newark, N. J.	June, 1899
White, A. C.	Hackensack, N. J.	April, 1893
White, F. H.	32 Nassau Street, New York City	April, 1884
White, G. M.	32 Nassau Street, New York City	April, 1886
*Whitwell, W. S.	Newark, N. J.	April, 1873
Wiener, I. K.	40 E. 41st Street, New York City	Dec., 1912
Wiesner, D. H.	40 E. 41st Street, New York City	Oct., 1880
*Wiggin, J.	Franklin Furnace, N. J.	April, 1881
Wilbur, F. P.	Franklin Furnace, N. J.	Dec., 1907
*Williams, H. R.	14 N. 3rd Street, Richmond, Va.	April, 1870
Williams, R. F.	(Died on Duty)	April, 1894
*Willis, C. N.	Lynchburg, Va.	April, 1883
Wilson, A. L.	405 West Chestnut Street, Louisville, Ky.	June, 1899
Wilson, F. C.	22 S. Hamilton Street, Poughkeepsie, N. Y.	April, 1869
Wilson, J. S.	5450 Germantown Avenue, Philadelphia, Pa.	Oct., 1888
Witherstone, C. S.	5509 Minerva Avenue, Chicago, Ill.	Oct., 1879
Woley, H. P.	United States Army	April, 1892
Wolfe, E. P.	Flushing, N. Y.	Dec., 1897
Wood, B.	133 Park Avenue, Mt. Vernon, N. Y.	Oct., 1879
Woodruff, W. S.	Tsing-Kiang-Pu, China.	Dec., 1907
Woods, J. B.	312 Haight Street, San Francisco, Cal.	April, 1892
Woodward, A. P.	China.	Oct., 1887
*Woolworth, E. W.	Ossining, N. Y.	Dec., 1898
Worth, G. B.	United States Army	Oct., 1894
Wren, R. J.	Babylon, N. Y.	June, 1907
Wyeth, M. C.	Yale, L. M.	April, 1880
Wynkoop, D. W.	128 E. 73rd Street, New York City	Dec., 1897
*Yale, L. M.	Riverhead, N. Y.	Oct., 1886
Yocum, J. G.		Sept., 1903
Young, H. H.		Oct., 1885

* Deceased.



